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# The Influence of Social and Economic Factors on Stillbirths and Neonatal Deaths

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IT has long been recognized that infant mortality is greatly influenced by social and economic factors. In depressed areas where there are slums the mortality is high and in prosperous areas it is low. In the same city and in any area, the mortality is low in high income groups (Registrar-General's Social Class I and II) and high in low income groups (Registrar-General's Social Classes III, IV, V).[1, 2] The social difference increases with the length of exposure to the unhealthy environment. Neonatal mortality in Class V is 50 per cent above that in Class I, but, in the age group 6 to 12 months, mortality in Class V is more than 4 times that in Class I.

It is known that in countries or areas where there is a high infant mortality, a high stillbirth-rate is also found. For example, in 1938 in Holland the stillbirth-rate was 25, the neonatal mortality-rate 21, and mortality in the period 1 to 12 months 15 per 1,000 live births. In England the corresponding figures were 38, 27 and 21, while in Scotland they were 42, 35 and 31. In the period 1939–41, the stillbirth-rate for Greater London was 30 per 1,000 total births and the infant mortality-rate 44, while during the same period the rates for Wales were 46 and 66. These figures suggest that stillbirths are affected in a general way by the same factors as infant mortality. There are very few records of analyses of stillbirth-rates by social class and the only one available for this country is that of the Registrar-General for Scotland for the year 1939,[3] when the stillbirth-rate for Scotland was 42.2 per 1,000 total births. On analysis the stillbirth-rates in the 5 social classes were 33.7, 37.8, 44.5, 38.0 and 42.3 respectively. The excess of the stillbirth-rate in Class V over that in Class I was 31.5 per cent, a much smaller social difference than is found with infant mortality.

On the other hand an analysis of maternal deaths by social class for the years 1930–32 in England and Wales showed that the maternal mortality-rate in Classes I and II was 4.44 per 1,000 births, an excess over the rate in Class V of 14.1 per cent. It might appear from these analyses that poor social and economic conditions, although they have an obvious and increasing effect on infant mortality after one month of age, have less effect on the neonatal mortality, still less on stillbirths and none at all on maternal mortality. The Report on a National Maternity Service by the Royal

College of Obstetricians and Gynaecologists[4] demonstrates clearly that, in fact, poor social conditions have a profound effect on maternal mortality. Similarly, further analysis of records of stillbirths and neonatal deaths suggests that poor social conditions have a serious effect on these death-rates also and that the relatively small difference in these rates between the different social classes is due to the interplay of factors which operate in opposite ways to affect mortality.

of pregnancy and many of this group are included in Group 1, since they were delivered in the same nursing home.

### Stillbirth-Rates.

Table I shows that the stillbirth-rate in Group 1, 25.4, is less than in Group 2, 30.4. When these rates are further analysed, we see that in Group 1 the component of the total stillbirth-rate due to death of full-time infants is 16.9 and that due to deaths of premature infants 8.5. In Group 2 the corres-

Table I.

*Analysis of Stillbirths and Neonatal Deaths in Three Groups of Cases in Aberdeen during the Years* 1938–44.

| | | Number | Percentage of total | Stillbirths | Stillbirth-rate | | Neonatal deaths | Neonatal mortality-rate | |
|---|---|---|---|---|---|---|---|---|---|
| Nursing Home Group: | | | | | | | | | |
| Group 1, 1,419 cases | Full-time | 1348 | 95.00 | 24 | 17.8 | 25.4 | 12 | 9.1 | 13.7 |
| | Premature | 71 | 5.00 | 12 | 169.0 | | 7 | 118.6 | |
| Booked Hospital Cases: | | | | | | | | | |
| Group 2, 8,808 cases | Full-time | 7999 | 90.82 | 132 | 16.5 | 30.4 | 131 | 16.7 | 34.5 |
| | Premature | 809 | 9.18 | 136 | 168.1 | | 164 | 243.7 | |
| Specialist Group: | | | | | | | | | |
| Group 3, 501 cases | Full-time | 481 | 96.01 | 4 | 8.3 | 10.0 | 3 | 6.3 | 8.1 |
| | Premature | 20 | 3.99 | 1 | 50.0 | | 1 | 52.6 | |

In order to compare stillbirth and neonatal mortality-rates in different social classes, the records of three groups of Aberdeen cases have been analysed: Group 1, a series of 1,419 delivered during the years 1938–44 in a nursing home where the expenses, apart from the doctor's fees were between 15 and 25 guineas, of whom some were under the care of specialists but the majority were looked after by the family doctor; Group 2, a series of 8,808 delivered in the Aberdeen Maternity Hospital as booked cases during the same 7 years at a cost per head of 2 pounds, all under the supervision of specialists; and Group 3, a series of 501 cases from private specialist practice. The last group was under the care of one specialist from the early months

ponding sub-rates are 15.0 and 15.4. This means that prematurity is a much more important factor in the causation of stillbirth in Group 2 than in Group 1. Although in both groups the stillbirth-rates in full-time and premature babies separately are practically the same, the total stillbirth-rate in Group 2 is higher than in Group 1 because the incidence of prematurity in Group 2 is almost double that in Group 1, and the stillbirth-rate in premature babies is 10 times the stillbirth-rate in full-time babies. In Group 3 the stillbirth-rate is only 10, due to the fact that not only is the incidence of prematurity less than in the other 2 groups, but the stillbirth-rate in both full-time and premature infants is much lower.

### Neonatal Mortality-Rates.

There is a much greater difference between Groups 1 and 2 in respect of neonatal mortality, the respective rates being 13.7 and 34.5. In contrast to stillbirths, the neonatal mortality-rates for both full-time and premature infants are much lower in Group 1 than in Group 2, being little more than half for full-time and less than half for premature infants. The much higher mortality-rate in premature infants in Group 2, combined with the greater incidence of prematurity in this group, accounts for much of the excess neonatal mortality in Group 2 over Group 1. The neonatal mortality-rate for Group 3 is 8.1 and the sub-rates for both full-time and premature infants are much lower than in either of the other groups.

Since there is no difference in the standard of obstetrical and nursing care between Groups 2 and 3, the difference in the stillbirth and neonatal mortality-rates is probably dependent on social status. If figures such as those quoted above are found generally, there is obviously room for great reduction in the stillbirth and neonatal mortality-rates in Scotland.

### Etiology of Stillbirths and Neonatal Deaths.

*Stillbirths.*

Postmortem examination may establish the immediate cause of foetal death but has only a limited value in indicating what measures are required to prevent stillbirths. For example, a postmortem examination may show that a child died of asphyxia or atelectasis. That explains how, not why the child died. The problem is all the more puzzling if the mother has been apparently well during the pregnancy and has had a normal labour.

In this series a postmortem examination was made of practically all cases in Group 2. The deaths have been classified according to the clinical condition of the mother except for foetal deformity, haemorrhagic disease and other foetal conditions incompatible with life. Premature stillbirths have been classified according to the cause of the prematurity. Prematurity is not in itself a cause of death, but since premature infants are particularly liable to injury and asphyxia (the stillbirth-rate in premature infants is 10 times, and the neonatal mortality-rate more than 10 times that in full-time infants), most progress in lowering stillbirth and neonatal mortality-rates will come from prevention of prematurity. Table II is an analysis of 268 stillbirths occurring in Group 2. It shows that in 34.8 per cent of full-time stillbirths, the cause of death could not be explained adequately; that is to say the child died *in utero* before the onset of labour, the foetal heart stopped during the course of a normal labour or the child was born with the heart still beating but failed to breathe. In respect of stillbirths in premature infants, the cause of the premature onset of labour was unknown in 33.8 per cent of the total.

Table III is an analysis of stillbirths in Group 1. It shows that the distribution of the causes of stillbirth is much the same as in Group 2 but, in full-time cases, there are fewer deaths from toxaemia and accidental haemorrhage and more from difficult labour. In Group 1 the proportion of elderly primiparae is higher than in Group 2, which would explain the higher proportion of stillbirths due to difficult labour and trauma. In Group 3, although the age and parity distribution is similar to that in Group 1, only 1 of the 5 stillbirths was associated with difficult labour. The number of cases in this group is small but it is interesting that Macafee[5] in a series of 424 cases in private specialist practice had only 5 stillbirths, of which one was due to cerebral haemorrhage. From the results of these 2 series one might expect a stillbirth-

TABLE II.

*Analysis of Causal Factors in 268 Stillbirths occurring in 8,808 "Booked" Cases in the Aberdeen Maternity Hospital during the Years 1938–44.*

GROUP 2.

| Full-time infants | | | Premature infants | | |
|---|---|---|---|---|---|
| Causal factor | Number | Percentage of total | Cause of premature onset of labour | Number | Percentage of total |
| Unknown ... ... ... | 46 | 34.8 | Unknown ... ... ... | 46 | 33.8 |
| Difficult labour ... ... | 41 | 31.1 | Eclamptic toxaemia ... | 29 | 21.3 |
| Eclamptic toxaemia ... | 18 | 13.6 | Foetal deformity ... ... | 19 | 14.0 |
| Accidental haemorrhage ... (Without toxaemia) | 10 | 7.6 | Accidental haemorrhage ... | 13 | 9.6 |
| | | | Intercurrent disease ... | 9 | 6.6 |
| Foetal deformity ... ... | 8 | 6.1 | Placenta praevia ... ... | 6 | 4.4 |
| Miscellaneous ... ... | 9 | 6.8 | Miscellaneous ... ... ... | 14 | 10.3 |
| Total ... ... ... | 132 | 100 | Total ... ... ... | 136 | 100 |

TABLE III.

*Analysis of Causal Factors in 36 Stillbirths occurring in 1,419 Nursing Home Cases during the Years 1938–44.*

GROUP 1.

| Full-time infants | | | Premature infants | | |
|---|---|---|---|---|---|
| Causal factor | Number | Percentage of total | Cause of premature onset of labour | Number | Percentage of total |
| Unknown ... ... ... | 10 | 41.7 | Unknown ... ... ... | 5 | 41.7 |
| Difficult labour ... ... | 11 | 45.8 | Eclamptic toxaemia ... | 4 | 33.3 |
| Eclamptic toxaemia ... | 2 | 8.3 | Foetal deformity ... ... | 1 | 8.3 |
| Accidental haemorrhage ... | — | — | Accidental haemorrhage ... | 2 | 16.7 |
| Foetal deformity ... ... | 1 | 4.2 | | | |
| Miscellaneous ... ... ... | — | — | | | |
| Total ... ... ... | 24 | 100 | Total ... ... ... | 12 | 100 |

rate of 10 with only 20 per cent of it due to trauma. In Group 1, the corresponding rate is 25.4 with 40 per cent of it due to trauma.

Conversely, as presumably avoidable factors such as trauma are eliminated by better obstetrical care, unavoidable causes such as foetal deformity account for a larger proportion of stillbirths. In Group 1 foetal deformity accounted for 4.2 per cent of the total stillbirths; in Group 3, combined with Macafee's series, it accounted for 40 per cent.

In the Registrar-General's analysis of causes of stillbirth in Scotland in the year 1939,[3] the total stillbirth-rate was 42.3 per 1,000 total births. It was found that "ill-defined" causes, e.g., asphyxia, debility, atelectasis, macerated foetus and prematurity, accounted for 23.4 per cent

of the total and the cause of the stillbirth was unknown in 9.5 per cent. Therefore in 32.9 per cent a satisfactory cause of stillbirth was not found. These findings are in keeping with the Aberdeen figures in the 3 groups under review.

labour is evident in the history of mortality in the highest social classes. Table IV, which is an analysis of the trend of mortality in nursing home cases in Aberdeen since 1933, shows a steady fall in the stillbirth-rate, so that in 1944 it is less than a

TABLE IV.

*Showing the Stillbirths and Neonatal Deaths at Different Dates in "Booked" Nursing Home Patients.*

(Rates per 1,000 in brackets)

| Date:— | 1933–37 | 1938–40 | 1941 | 1942 | 1943 | 1944 |
|---|---|---|---|---|---|---|
| Number of cases ... ... | 315 | 394 | 176 | 241 | 273 | 335 |
| Stillbirths ... ... | 15 (47.6) | 15 (38.1) | 5 (28.4) | 7 (29.0) | 6 (22.0) | 5 (14.9) |
| Neonatal deaths ... | 6 (20.0) | 4 (10.6) | 3 (17.5) | 3 (12.8) | 3 (11.2) | 4 (12.1) |

From the point of view of prevention of stillbirths an attempt was made in the Report on Infant Mortality in Scotland[6] to divide the cases in the Registrar-General's Report for 1939 into 2 groups, those arising primarily from the hazards of birth and those pre-existing in the child or mother. It was found that 38 per cent fell into the former group (hazards of birth) and 62 per cent into the latter group. When the cases in Group 2 in the present series are similarly analysed, it is found that 20 per cent of the stillbirths are in the category hazards of birth and 80 per cent in the category conditions pre-existing at birth. The total stillbirth-rate in Group 2 is 30.4 as compared to 42.3 for the whole country. Since the lower Aberdeen Maternity Hospital rate is due almost entirely to the lower proportion of stillbirths from hazards of birth, it appears as if the difference is to be attributed to better obstetrical technique during labour and not to superior health of the mothers during the antenatal period. If this is so, then there is obvious need for raising the general standard of obstetrical practice in Scotland.

The importance of careful routine antenatal care and skill in the management of

third of what is was in 1933–37. It might be argued that the improvement could be explained by advances in our knowledge of nutrition and improved diets for expectant mothers. It is true that these social classes are usually the first to take advantage of any advance in medical science. At the same time it is very doubtful if, at this economic level, there has been, so recently as 1933 or even earlier, any deficiency in nutrition sufficient to affect the stillbirth figures. It is much more likely that the improvement has been brought about mainly by better antenatal care and by improved technique during labour, with more frequent resort to specialist advice in abnormal cases. In Group 2 there appears to be little scope for further reduction in the stillbirth-rate by improved obstetrical care during the labour. Further improvement must come from better health of the mother before and during pregnancy.

## NEONATAL DEATHS.

In analysing the causes of neonatal mortality, the premature infants dying have been arranged according to the cause of the premature onset of labour, since, al-

though prematurity is not in itself the cause of death, the neonatal mortality-rate in premature infants is 10 times that in full-time. The most likely way to lower the neonatal mortality-rate, therefore, is to lessen the incidence of prematurity. This is further emphasized by the fact that in 75 per cent of these premature infants the immediate cause of death was weakness and atelectasis, and death occurred within a day or two of birth, so that there was little chance of preventing death even with the best medical and nursing care.

Table V is an analysis of 295 neonatal deaths in Group 2 and Table VI of 19 neonatal deaths in Group 1. In Group 2 30 per cent of deaths in full-time infants was due to infection, while in Group 1 there was no death from infection. Most of the deaths from infection in Group 2 occurred after the infants left hospital, an indication of the conditions under which they live. The

TABLE V.
*Analysis of Causal Factors in 295 Neonatal Deaths occurring in Hospital Cases.*
GROUP 2.

| Full-time infants | | | Premature infants | | |
|---|---|---|---|---|---|
| Cause of death | Number | Percentage of total | Cause of premature onset of labour | Number | Percentage of total |
| Infection ... ... ... | 40 | 30.6 | Unknown ... ... ... | 79 | 48.2 |
| Difficult labour ... ... | 33 | 25.2 | Eclamptic toxaemia ... ... | 21 | 12.8 |
| Foetal deformity ... ... | 30 | 22.9 | Twin pregnancy ... ... | 24 | 14.6 |
| Atelectasis or asphyxia ... | 7 | 5.3 | Accidental haemorrhage ... | 7 | 4.3 |
| Haemorrhagic disease ... | 5 | 3.8 | Placenta praevia ... ... | 12 | 7.3 |
| Unknown ... ... ... | 9 | 6.9 | Foetal deformity ... ... | 6 | 3.7 |
| Miscellaneous ... ... | 7 | 5.3 | Miscellaneous ... ... ... | 15 | 9.1 |
| Total ... ... ... | 131 | 100 | Total ... ... ... | 164 | 100 |

TABLE VI.
*Analysis of Causal Factors in 19 Neonatal Deaths occurring in 1,419 Nursing Home Cases.*
GROUP 1.

| Full-time infants | | | Premature infants | | |
|---|---|---|---|---|---|
| Causal factor | Number | Percentage of total | Cause of premature onset of labour | Number | Percentage of total |
| Infection ... ... ... | — | — | Unknown ... ... ... | — | — |
| Difficult labour ... ... | 4 | 33.3 | Eclamptic toxaemia ... | 2 | 28.6 |
| Atelectasis or asphyxia ... | 3 | 25.0 | Twin pregnancy ... ... | 1 | 14.3 |
| Foetal deformity ... ... | 3 | 25.0 | Accidental haemorrhage ... | 1 | 14.3 |
| Melaena ... ... ... ... | 1 | 8.3 | Placenta praevia ... ... | 1 | 14.3 |
| Erythroblastosis ... ... | 1 | 8.3 | Severe accident ... ... | 2 | 28.6 |
| Total ... ... ... | 12 | 100 | Total ... ... ... | 7 | 100 |

greatest difference between the 2 groups is in deaths of premature infants and the fact that, in 48 per cent of Group 2 cases, the cause of the premature onset of labour is unknown emphasizes the difficulty of preventing prematurity. In Group 1, 7 out of 12 deaths in full-time infants, 58 per cent, were due to difficult labour or asphyxia, occurred within 48 hours of birth and were related to the conduct of labour. Some of these deaths are avoidable. In Group 2 the corresponding percentage is 30, and in Group 3 not one of the 4 neonatal deaths was associated with difficult labour. Although the neonatal mortality-rate in Group 1 is very low, 13, it could be reduced still further by improved management of labour. In Group 2 the problem of neonatal mortality associated with difficult labour is complicated by the high incidence of contracted pelvis in this social class and its solution depends on the prevention of contracted pelvis as well as on improved management of labour.

Table VII is an analysis by age of 339 neonatal deaths occurring in the Aberdeen Maternity Hospital during the years 1941–44.

TABLE VII.

*Table showing an Analysis of 339 Neonatal Deaths in the Aberdeen Maternity Hospital during the Years 1941–44.*

| Day of death | | 1 | 2 | 3 | 4–7 | 2nd week | 3rd week | 4th week | Total | | Per cent |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Developmental malformations | Full-time | 4 | 3 | 2 | 3 | 2 | 3 | 0 | 17 | 22 | (8.0) |
| | Premature | 3 | 0 | 0 | 0 | 1 | 0 | 1 | 5 | | |
| Intracranial haemorrhage | Full-time | 17 | 8 | 3 | 1 | 2 | 0 | 0 | 31 | 46 | (16.7) |
| | Premature | 7 | 4 | 4 | 0 | 0 | 0 | 0 | 15 | | |
| Asphyxia | Full-time | 10 | 3 | 3 | 0 | 0 | 0 | 0 | 16 | 127 | (46.2) |
| | Premature | 79 | 13 | 6 | 10 | 3 | 0 | 0 | 111 | | |
| Infection | Full-time | 0 | 0 | 1 | 0 | 3 | 6 | 1 | 11 | 43 | (15.6) |
| | Premature | 0 | 0 | 0 | 3 | 12 | 11 | 6 | 32 | | |
| Miscellaneous | Full-time | 1 | 1 | 1 | 1 | 1 | 1 | 2 | 8 | 18 | (6.6) |
| | Premature | 3 | 1 | 2 | 3 | 1 | 0 | 0 | 10 | | |
| Inconclusive | Full-time | 0 | 0 | 1 | 1 | 1 | 0 | 1 | 4 | 19 | (6.9) |
| | Premature | 7 | 1 | 1 | 2 | 3 | 1 | 0 | 15 | | |
| Total | Full-time | 32 | 15 | 11 | 6 | 9 | 10 | 4 | 87 | 275 | |
| | Premature | 99 | 19 | 13 | 18 | 20 | 12 | 7 | 188 | | |
| No postmortem examination | Full-time | 6 | 2 | 0 | 1 | 5 | 1 | 1 | 16 | 64 | |
| | Premature | 23 | 11 | 4 | 4 | 4 | 2 | 0 | 48 | | |
| | | 160 | 47 | 28 | 29 | 38 | 25 | 12 | Total | 339 | |
| | | 235 (69 per cent) | | | | | | | | | |
| | | 264 (78 per cent) | | | | | | | | | |

44 and it shows that 235, or 69 per cent, died within 3 days of birth. Postmortem examinations of 275 were made. Of these 46.2 per cent died of asphyxia and these were, with a few exceptions, premature infants. The other common cause of death in the first 3 days was intracranial haemor-

rhage. After the 3rd day the most common cause of death was infection, with 15.6 per cent; 75 per cent of the infants dying of infection were premature and, without doubt, the prematurity predisposed to the infection, usually a pneumonia occurring in a partially expanded lung.

Any large maternity hospital dealing with great numbers of ill and premature babies requires a very large staff and well equipped nurseries to prevent the occurrence and spread of infection and to obtain the best results. In 1941 the Aberdeen Maternity Hospital enlarged its nursery for premature and ill babies and appointed a whole-time nursery staff and paediatric registrar in charge. During the 4 years since then, although the number of deliveries occurring in the hospital has increased from 1,300 to 1,600 per annum, the deaths of infants from infection have fallen as follows: 1941, 20; 1942, 10; 1943, 7; and 1944, 6. Even if all the deaths from infection were eliminated, there would still remain a large number of deaths due to other causes. Some of these can be prevented by the use of special boxes, in which conditions of temperature, humidity and oxygen pressure can be accurately controlled. Table VIII shows that of 234 premature infants dying, 186 died within a few days after birth. 132 of these weighed $3\frac{1}{2}$ pounds or less. These were born so prematurely that in most cases their organs had not reached a stage of development compatible with separate existence. The major problem, therefore, remains that of the prevention of prematurity.

In countries or cities where the infant mortality has fallen rapidly in recent years, the most striking improvement has been in the 1 to 12 months age group, so that now neonatal deaths form a much larger proportion of the total infant mortality. For example, in New York the infant mortality-rate was 57.3 in 1930 and in 1939 37.1, and Abramson[7] has shown that while the mortality-rate for the 1 to 12 months age group in 1930, 26.7, was almost equal to the neonatal mortality-rate, 30.6, the mortality in 1939 in the 1 to 12 months age group had fallen to half the neonatal, 12.3 and 24.8 respectively. He shows further that in premature infants, the fall in the mortality-rate from 14.0 to 11.6 is accounted for by a fall in the death-rate in the period after the first day. There has been a marked decline (63 per cent) in the 7 to 29 days period, some improvement (27 per cent) in the 2 to 7 days period, and none in the deaths occurring under 1 day.

The importance of these very early neo-

TABLE VIII.

*Cause of Neonatal Death in 234 Premature Infants in Aberdeen Maternity Hospital during the Years 1941–44.*

(Booked and emergency cases.)

| Birth weight in pounds | 5½–5 | 5–4½ | 4½–4 | 4–3½ | 3½–3 | Less than 3 | Total |
|---|---|---|---|---|---|---|---|
| Weakness or prematurity: lived less than 2 days or were feeble from birth | 11 | 20 | 18 | 22 | 27 | 88 | 186 |
| Infection ... ... ... ... ... | 3 | 5 | 8 | 5 | 7 | 9 | 37 |
| Erythroblastosis, foetal deformity and others ... ... ... ... ... | 8 | 0 | 1 | 1 | 1 | – | 11 |
| | 22 | 25 | 27 | 28 | 35 | 97 | 234 |

natal deaths is demonstrated by the following American figures.[8] In 1940 in the U.S.A., there were 67,866 neonatal deaths. Of these, 54,979 occurred under one week and 32,979 under one day. These figures agree with the findings in this paper that most of the neonatal deaths occur within a short time of birth and depend on the same factors as stillbirths, namely prematurity and feebleness of the baby, which we have shown to be associated with the social status of the mother.

There is much more information available about social class differences in neonatal mortality than there is for stillbirths. In 1930–32, according to the Registrar-General's analysis for England and Wales, the infant mortality-rate per 1,000 live births, attributed to premature birth, was 10.5 in Social Class I, and 19.6 in Class V, and that attributed to congenital debility 1.4 in Social Class I and 3.8 in Social Class V. When a country like Holland, where the neonatal mortality-rate is low (22 in the period 1932–36), is compared with Scotland, where the neonatal mortality-rate is high (37 for the same period), the excess mortality in Scotland is found to be in the category of prematurity and congenital debility. The figures for the 2 countries are as follows: in Holland, prematurity and congenital debility 9, congenital malformation and birth injury 7, other causes 6; in Scotland, prematurity and congenital debility 23, congenital malformation and birth injury 6, other causes 8. The neonatal mortality-rate in Holland at that period was the same as in Social Class I in England and Wales, while in Scotland the general rate was higher than in Social Class V in England and Wales. The excess of neonatal mortality of Scotland over that of Holland is due to prematurity and congenital debility, which also account for the excess neonatal mortality in Social Class V over Social Class I in England and Wales and are obviously associated with poor social and economic conditions, affecting adversely the physique, health and nutrition of the mother.

The neonatal mortality-rates obtained by Holland and Social Class I in England and Wales of 22 and 21.7 do not represent the lowest possible. In Group 1 of the Aberdeen series, the neonatal mortality-rate was 13.7, and in Group 3, 8.1. Macafee in his series had 4 neonatal deaths out of 426, 9.4 per 1,000, and Rietz[9] records a neonatal mortality-rate of 11.4 in Stockholm during the years 1918–22, in a series of cases in which the income per annum exceeded £600. It would appear that, where all social factors are favourable and medical and nursing standards good, the neonatal mortality-rate should not be more than 10.

## The Effect of Age and Parity on the Stillbirth and Neonatal and Mortality-Rates.

Table IX shows that the stillbirth-rate is relatively high with a first pregnancy, least in a second, and thereafter rises with each pregnancy. The rate is least in women under 25 years of age and in each parity it rises with age. Table X is a similar analysis of Group 2 in the Aberdeen series and it shows the same findings. When the age group under 25 is subdivided into those under 20 years of age and those between 20 and 25 years, the primiparae under 20 years of age show a stillbirth-rate of 17.7, compared to a rate of 21 in primiparae aged 20 to 24. The same conclusions are reached when a series of 5,820 cases delivered under the domiciliary scheme in the County of Aberdeen is studied. Here the total stillbirth-rate is lower than in Group 2, 25.2. The stillbirth-rate was 9.7 in primiparae under 20 years of age, and 28 in primiparae aged 20 to 24. These figures point to the extreme importance of youth

for safe childbirth, in fact they suggest that efficiency begins to diminish over the age of 20.

In the County of Aberdeen domiciliary series, in 327 women under 25 years, having a 3rd, 4th or 5th child, the stillbirth-rate was 6. In 364 similar cases in Group 2 women having a 3rd, 4th or 5th child, the mortality-rate in the age group 25 to 34 years is lower than in the age group 24 years and under (28.6 and 33.4 per cent respectively). This may be dependent more upon the deterioration of the environment and economic position of the mother in the

TABLE IX.

*Stillbirths (Legitimate) per 1,000 Children Born, by Age of Mother and Number of Previous Children*, 1938.

(Annual Report of Registrar-General for Scotland, 1939.)

| Number of previous children | All ages | Age of mother | | | | | |
|---|---|---|---|---|---|---|---|
| | | Under 20 | 20–24 | 25–29 | 30–34 | 35–39 | 40 and over |
| 0 | 49 | 29 | 37 | 48 | 62 | 98 | 95 |
| 1 | 30 | 32 | 21 | 29 | 35 | 46 | 38 |
| 2 | 32 | 19 | 16 | 27 | 45 | 42 | 60 |
| 3 | 41 | — | 27 | 31 | 44 | 58 | 80 |
| 4 | 43 | — | 35 | 25 | 45 | 61 | 60 |
| 5 | 43 | — | 25 | 30 | 38 | 52 | 72 |
| 6 | 53 | — | — | 35 | 38 | 66 | 78 |
| 7 | 59 | — | — | 30 | 33 | 68 | 91 |
| 8 | 58 | — | — | — | 48 | 49 | 96 |
| 9 | 61 | — | — | — | 26 | 66 | 82 |
| 10 and over | 86 | — | — | — | 30 | 106 | 90 |
| Total | 42 | 29 | 29 | 36 | 46 | 62 | 77 |

the rate was 13.7. In 1,371 women, aged 25 to 34 years in Group 2, having a 3rd, 4th or 5th child, however, the stillbirth-rate was 32.1. Despite the fact that in all probability the pregnancies would be better "spaced" in the older age group, the stillbirth-rate is more than double that in women under 25. Rapid childbearing up to a 4th or 5th does not seem to increase the risk for the child, provided the mother is young.

Table X shows an analysis of neonatal deaths similar to that of stillbirths. The neonatal mortality-rate varies with age and parity in much the same way as does the stillbirth-rate, although the variations are much less striking. The advantage of youth is much less obvious. In fact in younger age group, resulting from rapidly repeated pregnancy, than on deterioration of reproductive efficiency.

Table XI is a similar analysis of stillbirths in Group 1 and Group 3 cases. Unfortunately the age and parity were known only for the last 846 of Group 1, so that numbers are small when the cases are subdivided but in certain subdivisions they are large enough to allow valid comparisons to be made with Group 2. For example, in the age group 25 to 34, in Group 3, there was no stillbirth in 199 cases; in Group 1 the stillbirth-rate was 11.7 and in Group 2, 53.4. The total stillbirth-rate in primiparae in Group 2 is 44 per cent in excess of that in Group 1 and 236 per cent in excess of that in Group 3, despite the fact that

TABLE X.

*Stillbirth-rates and Neonatal Mortality by Age and Parity of Mother.*
*Booked Hospital Cases: Group 2.*

Stillbirths

| Parity | Under 25 | | | 25–34 | | | 35+ | | | All ages | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate |
| 1 | 2431 | 49 | 20.2 | 1237 | 66 | 53.4 | 168 | 14 | 83.3 | 3836 | 129 | 33.6 |
| 2 | 858 | 11 | 12.8 | 1135 | 23 | 20.3 | 152 | 9 | 59.2 | 2145 | 43 | 20.0 |
| 3, 4, 5 | 364 | 5 | 13.7 | 1371 | 44 | 32.1 | 361 | 12 | 33.2 | 2096 | 61 | 29.1 |
| 6, 7, 8 | 8 | 0 | 0 | 296 | 9 | 30.4 | 241 | 12 | 49.8 | 545 | 21 | 38.5 |
| 9+ | — | — | — | 38 | 4 | 105.3 | 148 | 10 | 67.6 | 186 | 14 | 75.3 |
| All parities | 3661 | 65 | 17.7 | 4077 | 146 | 35.8 | 1070 | 57 | 53.3 | 8808 | 268 | 30.4 |

Neonatal mortality

| Parity | Under 25 | | | 25–34 | | | 35+ | | | All ages | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate |
| 1 | 2382 | 78 | 32.7 | 1171 | 53 | 45.3 | 154 | 6 | 39.0 | 3707 | 137 | 37.0 |
| 2 | 847 | 25 | 29.5 | 1112 | 29 | 26.1 | 143 | 7 | 48.9 | 2102 | 61 | 29.0 |
| 3, 4, 5 | 359 | 12 | 33.4 | 1327 | 38 | 28.6 | 349 | 14 | 40.1 | 2035 | 64 | 31.4 |
| 6, 7, 8 | 8 | — | — | 287 | 14 | 48.8 | 229 | 12 | 52.4 | 524 | 26 | 49.6 |
| 9+ | — | — | — | 34 | 2 | 58.8 | 138 | 5 | 36.2 | 172 | 7 | 40.7 |
| All parities | 3596 | 115 | 32.0 | 3931 | 136 | 34.6 | 1013 | 44 | 43.4 | 8540 | 295 | 34.5 |

| | |
|---|---|
| 1st pregnancy ... ... | 43.6 per cent |
| 2nd pregnancy ... ... | 24.3 ,, ,, |
| 3rd, 4th and 5th pregnancy | 23.8 ,, ,, |
| 6th + pregnancy ... ... | 8.3 ,, ,, |

TABLE XI.

*Analysis of Stillbirth-rates and Neonatal Mortality in Nursing Home: Group 1 and Specialist Cases: Group 3 by Age and Parity of Mother.*

Nursing Home: Group 1.

Stillbirths

| Parity | Under 25 | | | 25–34 | | | 35+ | | | All ages | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate |
| 1 | 102 | 3 | 29.4 | 341 | 4 | 11.7 | 73 | 5 | 68.5 | 516 | 12 | 23.3 |
| 2 | 17 | 1 | 58.8 | 166 | 2 | 12.0 | 65 | 1 | 15.4 | 248 | 4 | 16.1 |
| 3, 4, 5 | | | | 32 | 0 | 0 | 49 | 2 | 40.8 | 81 | 2 | 24.7 |
| 6+ | | | | | | | 1 | 0 | 0 | 1 | 0 | 0 |
| All parities | 119 | 4 | 33.6 | 539 | 6 | 11.1 | 188 | 8 | 42.5 | 846 | 18 | 21.3 |

Neonatal mortality

| Parity | Under 25 | | | 35+ | | | 35+ | | | All ages | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate |
| 1 | 99 | 1 | 10.1 | 337 | 5 | 14.8 | 68 | 0 | 0 | 504 | 6 | 11.9 |
| 2 | 16 | 0 | 0 | 164 | 1 | 6.1 | 64 | 1 | 15.6 | 244 | 2 | 8.2 |
| 3, 4, 5+ | | | | 32 | 0 | 0 | 48 | 0 | 0 | 80 | 0 | 0 |
| All parities | 115 | 1 | 8.7 | 533 | 6 | 11.3 | 180 | 1 | 5.6 | 826 | 8 | 9.7 |

1st pregnancy ... ... 61.0 per cent
2nd pregnancy ... ... 29.3 ,, ,,
3rd, 4th and 5th pregnancy 9.6 ,, ,,

TABLE XI (*Continued*).

Specialist Cases: Group 3.

Stillbirths

| Parity | Under 25 | | | 25–34 | | | 35+ | | | All ages | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate | Number of births | Stillbirths | Rate |
| 1 | 56 | 2 | 35.7 | 199 | 0 | 0 | 45 | 1 | 22.2 | 300 | 3 | 10.0 |
| 2 | 9 | 0 | 0 | 85 | 0 | 0 | 29 | 0 | 0 | 123 | 0 | 0 |
| 3, 4, 5 | 1 | 0 | 0 | 48 | 1 | 20.8 | 29 | 1 | 34.5 | 78 | 2 | 25.6 |
| All parities | 66 | 2 | 30.3 | 332 | 1 | 3.0 | 103 | 2 | 19.4 | 501 | 5 | 10.0 |

Neonatal mortality

| Parity | Under 25 | | | 25–34 | | | 35+ | | | All ages | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate | Number of live births | Neonatal deaths | Mortality-rate |
| 1 | 54 | 0 | 0 | 199 | 3 | 15.1 | 44 | 0 | 0 | 297 | 3 | 10.1 |
| 2 | 9 | 0 | 0 | 85 | 0 | 0 | 29 | 0 | 0 | 123 | 0 | 0 |
| 3, 4, 5+ | 1 | 0 | 0 | 47 | 1 | 21.3 | 28 | 0 | 0 | 76 | 1 | 13.2 |
| All parities | 64 | 0 | 0 | 331 | 4 | 12.1 | 101 | 0 | 0 | 496 | 4 | 8.1 |

1st pregnancy ... ... 59.9 per cent
2nd pregnancy ... ... 24.5 ,, ,,
3rd, 4th and 5th pregnancy 15.6 ,, ,,

age and parity distribution, as appears from Table IX of national figures, is greatly in favour of a stillbirth-rate lower in Group 2 than in either of the other groups. In Group 2, 63.4 per cent of the primiparae are under 25 years of age; in Group 1 only 19.8 per cent are in this age group, and in Group 3, 18.7 per cent. Since the standard of medical and nursing care is the same in Groups 2 and 3, the excess stillbirth-rate in primiparae (236 per cent) of Group 2 over Group 3 must be due to social and economic factors. The excess is minimized by the favourable age distribution in Group 2 (the percentages of all primiparae in Group 2, in the age groups, under 25, 25 to 34 and 35 and over are 63.4, 32.2 and 4.4 respectively and in Group 3, 18.7, 66.3 and 15.0 respectively). If Group 2 had the same large percentage as Group 3 of primiparae in the 25 to 34 age group, the excess of the stillbirth-rate of Group 2 over Group 3 would obviously be greatly increased since, in this age group, the stillbirth-rate in Group 2 is 5 times that in Group 1.

As regards the effect of high parity in Group 2, 8.3 per cent have had more than 5 previous pregnancies and in these the stillbirth-rate is 47.9. There is only one case in this category in Groups 1 and 3.

It has been shown in the Report on Infant Mortality in Scotland (1943) that despite unfavourable age and parity distribution, certain areas may have a low stillbirth-rate. Greater London, with a worse-than-average age and parity distribution has the lowest stillbirth-rate of all the large cities in England and Wales. In Scotland the age and parity distribution is more favourable to a low stillbirth-rate than in any of the English areas but its actual rate is higher than any except North IV and the 2 Welsh areas. The 2 worst areas in England and Wales, North IV and Wales I show an excess stillbirth-rate at all ages. In the age groups 20 to 24, the excess over London is 50 per cent. This cannot be attributed to closeness of the spacing of births and consequent higher average parity, because the general distribution shows no definite increase, even with the 4th birth in this age group. Hence there are influences operating in Wales and the North of England which increase the stillbirth-rate even between the ages of 20 and 25, when the prospects are best. Since these are depressed areas, the influences most likely to be responsible are poor physique of the mother, poor diet during pregnancy and probably a poor standard of obstetric care.

To discover why the stillbirth-rate rises after the age of 20 and still more after the age of 25 in Group 2, the stillbirths and neonatal deaths in Group 2 have been analysed in detail and the results are shown in Table XII. Neonatal deaths have been included with stillbirths here as they are, apart from infection, due to the same causes. Under the heading "cause unknown" are included all cases in which the death could not be explained adequately by a complication of pregnancy or labour, or, in the case of premature infants, when the cause of prematurity was unknown. When a premature child survived more than 2 days and died of sepsis, the death is attributed to infection, even although the prematurity was obviously an important factor in predisposing to infection. In primiparae the most important class is "cause unknown," followed by trauma, toxaemia, foetal deformity and sepsis. With increasing age of primiparae, the mortality from toxaemia, "cause unknown" and trauma rises, there is no significant rise in the death-rate from infection and a slight fall in the mortality from foetal deformity. The same is true of the second pregnancy but thereafter the figures are much more variable, since in the younger age groups of the same parity as older age groups the more rapid

TABLE XII.

*Mortality Rates (Stillbirth and Neonatal Together) by Cause, Age of Mother and Order of Birth.*

Booked Hospital Cases: Group 2.

| Age of mother | Birth I. Under 25 | 25–34 | 35+ | All ages | Birth II. Under 25 | 25–34 | 35+ | All ages |
|---|---|---|---|---|---|---|---|---|
| Number of cases | 2431 | 1237 | 168 | 3836 | 858 | 1135 | 152 | 2145 |
| Cause of death: | | | | | | | | |
| Cause unknown ... ... | 1.35 | 3.03 | 4.7 | 2.06 | 1.63 | 1.41 | 2.63 | 1.58 |
| Toxaemia ... ... ... | 0.65 | 1.13 | 2.9 | 0.91 | 0.23 | 0.88 | 1.31 | 0.65 |
| Trauma ... ... ... ... | 0.98 | 1.53 | 2.9 | 1.24 | 0.34 | 0.44 | 1.9 | 0.51 |
| Foetal deformity ... ... | 0.90 | 0.88 | 0.6 | 0.88 | 0.23 | 0.61 | 0.65 | 0.46 |
| Infection ... ... ... | 0.70 | 0.88 | 1.1 | 0.78 | 0.34 | 0.61 | 1.9 | 0.55 |
| Haemorrhagic disease ... | — | — | — | 0.10 | — | — | — | 0.09 |
| Placenta praevia ... ... | — | — | — | 0.07 | — | — | — | 0.14 |
| Accidental haemorrhage (without toxaemia) ... | 0.21 | 0.72 | — | 0.36 | 0.11 | 0.17 | — | 0.14 |
| Miscellaneous ... ... ... | 0.28 | 0.97 | 0.6 | 0.52 | — | — | — | 0.65 |

| Age of mother | Birth III, IV and V. Under 25 | 25–34 | 35+ | All ages | Birth VI and over. Under 25 | 25–34 | 35+ | All ages |
|---|---|---|---|---|---|---|---|---|
| Number of cases | 364 | 1371 | 361 | 2096 | 8 | 334 | 389 | 731 |
| Cause of death: | | | | | | | | |
| Cause unknown ... ... | 2.20 | 2.26 | 1.94 | 2.19 | — | 3.29 (11) | 2.57 (10) | 2.87 (21) |
| Toxaemia ... ... ... | 0.27 (1) | 0.80 | 0.55 (2) | 0.67 | — | — | 1.03 (4) | 0.55 (4) |
| Trauma ... ... ... ... | — | 0.36 (5) | 0.55 (2) | 0.33 | — | 0.60 (2) | 1.80 (7) | 1.23 (9) |
| Foetal deformity ... ... | 0.27 (1) | 0.51 | 1.66 | 0.67 | — | — | 1.29 (5) | 0.68 (5) |
| Infection ... ... ... | 0.82 (3) | 0.80 | 0.28 (1) | 0.72 | — | 0.30 (1) | 1.03 (4) | 0.68 (5) |
| Haemorrhagic disease ... | 0.27 (1) | 0.21 (3) | 0.28 (1) | 0.24 | — | 0.90 (3) | — | 0.41 (3) |
| Placenta praevia ... ... | — | 0.36 (5) | 0.28 (1) | 0.29 | — | 1.20 (4) | 0.77 (3) | 0.96 (7) |
| Accidental haemorrhage (without toxaemia) ... | — | 0.29 (4) | 0.83 (3) | 0.33 | — | 2.10 (7) | 0.51 (2) | 1.23 (9) |
| Miscellaneous ... ... ... | 0.82 (3) | 0.36 (5) | 0.83 (3) | 0.52 | — | 0.30 (1) | 1.03 (4) | 0.68 (5) |

succession of pregnancies has led to deterioration.

The figures for all ages together show that the combined stillbirth and neonatal mortality from toxaemia is highest in a 1st pregnancy but after the 2nd remains fairly steady. Mortality of "causes unknown" is relatively high in a 1st pregnancy, falls in a 2nd and rises steadily thereafter. As would be expected, that due to trauma is high in the 1st pregnancy, falls in the 2nd, 3rd, 4th and 5th, and thereafter rises. This rise in the high parity group is associated with malposition, such as breech presentation and prolapse of the umbilical cord. There is little change with increasing parity in the death-rate from deformity or infection. Haemorrhagic diseases are a very small group in a 1st pregnancy but become a more important cause of death with increasing parity. Placenta praevia as a cause of death rises steadily with increasing parity, as also does accidental haemorrhage without toxaemia

The analysis shows that the increase in the combined stillbirth- and neonatal death-rates with the age of the mother in Group 2, especially in primiparae, is due to increased mortality from toxaemia, "cause unknown" and trauma. "Cause unknown" is the most important factor. This means in effect that with increasing age of the mother there are more premature babies and more feeble full-time babies, who die *in utero* before the onset of labour or who cannot stand the strain of even a normal labour. The increased mortality from trauma is explained by the well-known increase with age of prolonged and difficult labour. It is difficult to say whether disordered uterine action as a cause of difficult labour is more common at this economic level than higher in the social scale but the foetal mortality from this cause will almost certainly be higher because of the higher proportion of premature and feeble babies. Prolonged labour from contracted pelvis is more common in the lower income group.

Numbers in Groups 1 and 3 are too small to permit of similar analysis but it is noteworthy that the total foetal mortality per cent in primiparae in the age group 25 to 34 in Group 1 is 2.65 per cent and in Group 2, 9.87. In Group 2 the mortality due to "cause unknown" is 3.03 in this age group, more than the total from all causes for the same age group in Group 1. The main cause of the excess mortality in Group 2 is the high proportion of premature and feeble babies. The capacity to produce strong and healthy babies appears to fall off rapidly in primiparae in Group 2 after the age of 20, and it is only because 66 per cent of the primiparae are under 25 years of age in Group 2 that the excess mortality over Group 1 is not very much greater. In Group 1 primiparae, on the other hand, the mortality remains low till after the age of 34. In a paper to follow, it will be shown that the women in Groups 1 and 3 are much taller than those in Group 2 and the babies heavier. There is less contracted pelvis and, although accurate criteria of the nutritional state are not available, general examination shows great differences between the two groups in the state of the teeth and gums, the texture of the skin and hair and general well-being. These appearances leave no doubt that the state of health and nutrition in Groups 1 and 3 is much superior to that in Group 2.

The problems involved in lowering the stillbirth- and neonatal death-rates are different for Groups 1 and 2. In Group 1 a high standard of care during labour must be ensured since most deaths are due to trauma, dependent on the fact that a high proportion of the women are elderly primiparae. The danger run by this group despite its favourable economic position is shown by the high stillbirth-rate of 33.7 for

Social Class I in Scotland in 1939.[3] As a long-term policy measures should be introduced to encourage women in this social class to have their first baby at an earlier age. In Group 2 the primary need is to improve social and economic conditions and so raise the standard of nutrition and general health. This would result in less prematurity and fewer feeble babies. Contraceptive advice should be made more easily available to this group, as the stillbirth-rate is high after the fifth pregnancy, and few of these women wish to have more than 5 children. Since, in Scotland, about 90 per cent of the mothers are in lower income groups corresponding to Group 2 in this series, the biggest reduction in Scotland's stillbirth- and neonatal mortality-rates will result from improvement in social and economic conditions.

## Summary.

1. In order to compare stillbirth- and neonatal mortality-rates in different social classes, the records of 3 groups of Aberdeen cases have been analysed: Group 1, a series of 1,419 delivered in a nursing home, belonging to the Registrar-General's social classes I and II and mostly under the care of the family doctor; Group 2, a series of 8,808 booked hospital cases, under the care of specialists, belonging to social classes III, IV and V; Group 3, 501 cases in private specialist practice. In the 3 groups the stillbirth-rates were 25.3, 30.4 and 10.0 respectively, and the neonatal mortality 13.0, 34.5 and 8.1.

2. In Groups 1 and 2 the stillbirth-rates in full-time and premature infants were the same, and in each group the stillbirth-rate in premature infants was 10 times that in full-time infants. The excess mortality of Group 2 over Group 1 was due to the incidence of prematurity in Group 2 being almost double that in Group 1.

3. The patients in Groups 1 and 3 are in the same social class, and the differences in the stillbirth- and neonatal mortality-rates are probably due to different standards of obstetric care. In Group 1 the stillbirth-rate has fallen from 47.6 in the years 1933–37, to 14.9 in the year 1944. This fall is due mainly to improved obstetrics.

4. In Group 2 the stillbirth-rate is 3 times that in Group 3, although the standard of obstetrics is the same. There is very little scope for reduction in the stillbirth-rate in Group 2, except by measures designed to improve the health and nutrition of the mother.

5. The problem of the high neonatal mortality in Group 2 is largely one of the prevention of prematurity. Seventy per cent of the deaths in premature infants occurred within 48 hours of birth, most of them being too feeble to maintain a separate existence.

6. The stillbirth-rate is relatively high with first pregnancies, least in the 2nd and thereafter rises with each pregnancy. The rate rises with age in each parity. In Group 2 the stillbirth-rate in the age group 25 to 34 in primiparae is nearly 5 times that in the same age group of primiparae in Group 1.

7. The reproductive efficiency in Group 2 as measured by the stillbirth-rate falls off steadily after the age of 20, whereas in Groups 1 and 3 the fall in efficiency is delayed till the age of 30.

8. In Group 2, 63 per cent of the primiparae are under 25 years of age and in Groups 1 and 3, 19 per cent. The stillbirth-rate in the latter groups would be very high if the reproductive efficiency in these groups fell off as quickly with age as it does in Group 2.

9. Over 30 per cent of the stillbirths in full-time infants in Group 2 are due to intrauterine death of the foetus from unex-

plained causes. The cause of the onset of premature labour is unexplained in about 50 per cent of cases in Group 2. The most probable explanation in both cases is poor health and nutrition of the mother.

10. From a national point of view, the stillbirth- and neonatal mortality-rates will be most substantially improved by improvement in the standard of health and nutrition of the mothers in Social Classes III, IV and V, corresponding to Group 2 in this series, as they constitute the vast majority.

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# A Case of Arrhenoblastoma Complicating Pregnancy

BY

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THE case here described appears to be unique, for although some 60 cases of this type of tumour have been recorded, not one has occurred associated with pregnancy.

Mrs. F., 26 years of age, was a woman whose previous medical history was without particular interest. Six months before she became pregnant her appendix was removed, but no comment was made as to the condition of the pelvic organs. Three months later she suffered from vomiting for several days for which a cause was not found. She menstruated quite normally on December 14th, 1942, and the pregnancy began during the next menstrual cycle. In the following February it was found that she had glycosuria, and without complete investigation her carbohydrate intake was very severely reduced. In April she noticed that hair was growing on her abdomen, and from there spread to her chest and face, and also became luxuriant on her arms and legs. Her skin became coarse, acne developed on her face and shoulders, and this was followed by crops of boils over the area affected by the acne. About the time that she noticed hair on her face her friends began to remark on her voice, which had become deeper than before. It was impossible to discover any psychological alteration. She could offer no opinion on her sexual feelings or reflexes; her reply to a direct question was that her husband had been away from home in the forces for the past few months. She thought she had lost weight but could produce no evidence for the opinion.

She came to the out-patient department of the Saint Mary's Hospitals, Manchester, at the end of July. She then had a masculine distribution of hair, a masculine voice and figure, except that the abdomen was distended by a 7 months' pregnant uterus, and the breasts were enlarged and showed all the usual signs of pregnancy. The foetus presented by the breech, and X-ray examination showed that the legs were extended.

The clitoris was much enlarged, but there was no alteration in the labia majora or minora. Vaginal examination showed the presence of a tumour with smooth surface, separate from the uterus, about the size of a Jaffa orange, situated in the pelvis and pushing the cervix forwards into close relation with the pubes and preventing descent of the foetal breech.

She was admitted to hospital a few days later, and remained there for 2 months. She did not show any desire to return home before delivery, probably on account of her somewhat repulsive appearance.

Although the glycosuria remained constant until 3 weeks after delivery, estimation of her blood sugar on many occasions showed that it was within normal limits. On her arrival at hospital after 5 months of severe reduction of carbohydrate intake, in addition to sugar, acetone and diacetic acid were present in her urine, but estimation of her blood sugar showed that it was

94 mg. per cent. When her diet became normal the percentage rose to 125. It is clear that she was not a true diabetic; nor was she a typical example of the glycosuria of pregnancy, for in the latter the sugar comes and goes in the urine like a will o' the wisp: it is there one day and gone the next without reason. This would appear to be an example of the "Achard-Tiers syndrome," sometimes referred to as "diabetes of bearded women." I know of no satisfactory explanation of the phenomenon.

At this time she was very anaemic, her red blood corpuscle count being 3,790,000, and the haemoglobin 55 per cent of normal. The gloom of Manchester could not be offered as a partial explanation of this anaemia for she lived some 20 miles distant, in Cheshire. X-ray of the skull showed an unusually large dorsum sellae. The Aschheim-Zondek test was positive.

Owing to the extreme efficiency of the nursing staff the lower abdomen was shaved within a few hours of admission, and the photographs were taken a fortnight later. Although they fail to show the original luxuriant growth of hair on the abdomen, they do show how rapidly it could grow again (Figs. 1, 2, 3, 4).

Her waters broke prematurely at the 37th week. There were no symptoms or signs of labour starting, but it was decided that Caesarean section should be performed at once. Moderate pressure had failed to dislodge the obstructing tumour from the pelvis, and greater force was not considered justifiable. The operation was through the upper uterine segment under local anaesthesia following morphia and scopolamine premedication. The tumour proved to be a new growth of the right ovary and was removed. The left ovary was normal with the usual rusty appearance of its surface.

Lactation began on the 4th day after delivery and was established satisfactorily. Involution of the uterus and the lochial discharge were about normal for a Caesarean section case.

The patient was seen 3 months after delivery, and it was found that although there was no change in her voice, yet the masculine distribution of hair was gone except for a suggestion of a moustache. The septic skin condition had continued, and her face and chest were badly scarred. There was further improvement again 3 months later; the child was still breast-fed.

There is no doubt that this is a typical case of arrhenoblastoma with characteristic symptoms and signs except where these were interfered with by the pregnancy. The reason for the extreme rarity of the combination of the two conditions is that once the tumour has produced its first symptoms, amenorrhoea, the woman is sterile, but usually several months must pass before the symptoms of virilism appear. There is, therefore, only a very short period during which pregnancy and virilism can synchronize.

Novak stresses the fact that the first symptoms of the tumour are defeminizing, amenorrhoea and atrophy of the breasts. This case showed neither of these symptoms. The amenorrhoea in her case was of pregnancy, and there was no atrophy of the breasts. It is agreed that the defeminizing phenomena are caused by the failure of the ovaries to secrete oestrin. This cessation of ovarian function is in its turn ascribed to the male hormone causing the pituitary to fail to secrete its gonadotrophic hormone. The increased secretion of both ovarian hormones from some source other than the ovary, which occurs in normal pregnancy, must have occurred in this pregnancy also, and been the cause of the enlargement and later the glandular activity of the breasts.

These new growths are described as of slow growth. Without doubt the increased vascularity of the pelvis during pregnancy

C P.B.

PLATE I.

Fig. 1.

Mrs. F., 34th week of pregnancy, showing masculinization of figure.

C.P.B.

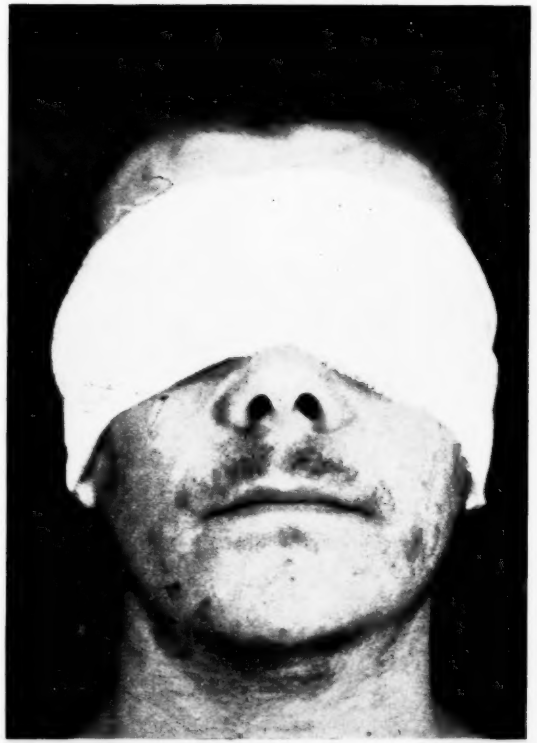

Fig. 2.
Showing growth of hair on face.

FIG. 3.

Showing normal pregnancy changes in breasts and the growth of hair on upper abdomen and chest; also acne on chest.

FIG. 4.

Showing the enlarged clitoris and the fortnight's growth of hair on the abdomen.

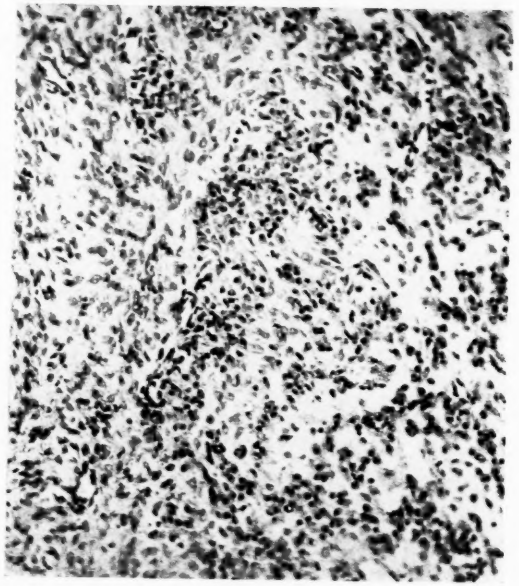

FIG. 5.

Section of tumour showing on the left one of the fibrous bands with which the growth is intersected and, on the right, a cellular area. × 112

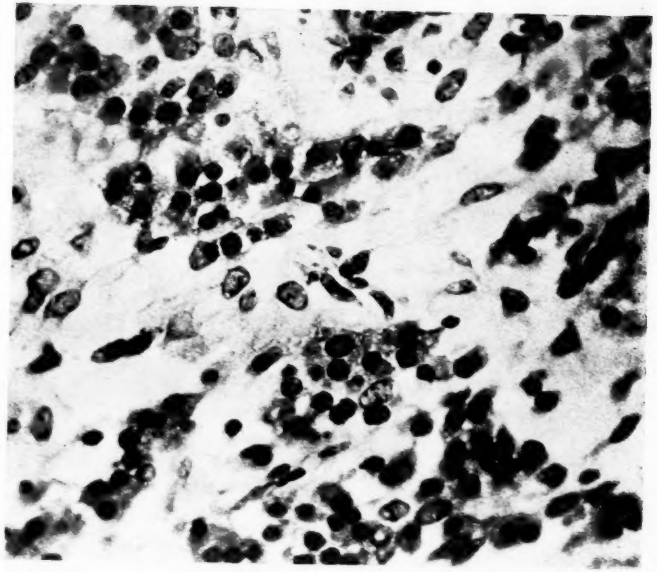

FIG. 6.

High-power view of one of the cellular areas, showing collections of cells resembling Leydig cells in a fibrous matrix. × 370

FIG. 7.
Female child of Mrs. F.

FIG. 8.

Child, showing enlarged clitoris, no labia minora, urethra seen with difficulty at the base of the clitoris.

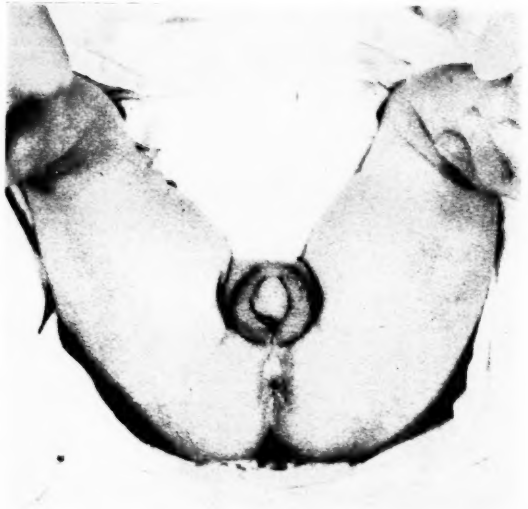

FIG. 9.

Child, showing enlarged clitoris, labia majora, enlarged and corrugated; no labia minora. Urethral opening cannot be seen.

would accelerate the rate of growth of this tumour. Judging from its size when removed in August it is justifiable to assume that it had been present for at least 12 months. From the fact that sufficient male hormone was secreted in the spring to produce masculine changes in the mother in April, it may also be assumed that a lesser quantity was being secreted at the end of the previous year, but not sufficient to interfere with the function of the pituitary gland. The quantity of the male hormone increased during the months of January and February, until in March sufficient was produced to cause the appearance of virilism in the mother in April. Usually these latter phenomena are delayed for a much longer period than this.

The tumour was of a dirty greenish colour, with a smooth tough outer coat, and in size was $3\frac{1}{2}$ by 3 by 2 inches (Plate A). It was irregularly lobulated, and its consistence varied, about one third being very hard and the remainder soft. When the growth was bisected it was clear that the hard area was a fibroma; the remainder was soft and in places cystic, somewhat resembling a degenerated fibroid, but having a pale greenish colour.

Professor J. H. Dible who examined sections of the tumour reports: "I have examined a whole section of this growth having an area of about 30 sq. cm. The tumour shows bands of well developed cellular fibrous tissue, showing well staining collagen fibres, which surround other areas made up of polygonal cells having rather an epithelial appearance scattered diffusely in a fibrous matrix. Where the latter is most developed the cells are compressed, but where it is more open or oedematous these show as groups of polyhedral cells which have abundant granular eosinophilic cytoplasm and a relatively small dark-staining nucleus: these cells resemble the interstitial (Leydig) cells of the testicle. Fig. 5 shows these two separate appearances of the growth and Fig. 6 shows typical islets of Leydig cells.

Ovarian tumours associated with virilism are not of a single histological type and the term arrhenoblastoma has been criticized by a number of writers since it is used to suggest a functional activity of the growth and does not describe its morphology or histogenesis. For these reasons Burrows[1] prefers the word "arrhenoma," which is not open to this objection. The majority of such tumours of the ovary fall into one or other of the following histological groups.

(*a*) Growths showing structures frankly resembling testicular tubules (Pick's *adenoma tubulare testiculare ovarii*).

(*b*) Those showing epithelial cords without lumina, which are believed to represent the solid cords of the developing *rete testis*.

(*c*) Those consisting largely of cellular fibromatous tissue which is sometimes so cellular as to be described as sarcomatous.

(*d*) Teratomatous growths which may show in certain areas the development of male gonadic tissue.

Interstitial testicular (Leydig) cells are often found in the less differentiated forms, but have not been constantly reported in the tubular type of tumour. In addition, adrenal cortical tumours in the ovary may occur and produce virilism.

The growth in the present case is in the group (c) above, the members of which do not show recognizable tubular elements, but have an abundance of Leydig cells and large areas of fibromatous stroma. Attempts to demonstrate sudanophilic or lipoid material in these cells in frozen sections were not very successful: a few cells showed some fatty granules, but as the only material available at this stage had been

passed through spirit, for colour preservation, no significance can be attached to this failure."

The child, which weighed 6½ pounds, appeared normal except for the external genital organs (Figs. 7, 8, 9). It may be said at once that it is almost certainly a female pseudohermaphrodite, although thorough investigation of its genito-urinary tract must obviously be postponed for several years. The clitoris was much enlarged and the glans, prepuce and frenum were well formed. The urethra opened at the base of the clitoris and behind this the perineum extended unbroken to the anus. A median raphe separated the 2 labia majora, which were enlarged and corrugated. The labia minora were absent as such. On the 4th day of life there was a discharge of blood and mucus from the urethra such as occasionally occurs from the vagina of newborn girls of this age and is usually attributed to the sudden withdrawal of maternal oestrin from the child's endometrium stimulated by that hormone. This haemorrhage was strong presumptive evidence of the presence of a uterus and a vagina opening into the urethra; and therefore of the child being a female pseudohermaphrodite.

When the mother brought the child to hospital 3 months after delivery, she reported that the haemorrhage had recurred when the child was about 4 weeks old, and again when about 8 weeks old. I have been unable to think of any possible explanation of this strange phenomenon. There has not been any similar haemorrhage since.

One naturally asks how this variety of female pseudohermaphrodite develops. There is no doubt that normally the growth or atrophy of the sex ducts, of which both male and female are provisionally laid down in the early stage of development, and the form taken by the external genital organs, including the urogenital sinus, depend on the recently determined sex of the gonad. Although nature has made many experiments of administering male hormones to the female human mammal, with one exception all these have been adults. Nature's only experiment on the human foetus, namely the adrenogenital syndrome beginning in early foetal life, has resulted at times in a condition similar to the one here described.

The best-known of nature's experiments on mammals results in the freemartin. Here is an extraordinary and inexplicable mixture of phenomena: gonads that approach the male form, male gonoducts, but external genitalia of the female type. In the light of laboratory experiments one would have expected all the accessory genital organs to have been definitely male. Wiesner[2] in propounding his monhormonic theory of development suggests that this does in fact occur at times, but, because the animal is so typically male in external appearance, it is never recognized that it is really a freemartin. The evidence is against this theory, for when the freemartin is of triplets, the other 2 being male, and the concentration of male hormone is therefore doubled, its external genitalia still retain the female form.

Much experimental work has been done to investigate the results of treating pregnant mammals with androgens, Raynaud[3] on mice, Dantchakoff[4] on guinea-pigs, and Greene[5] on rats. Greene points out that results naturally depend on 2 factors, the quantity of male hormone administered, and the period of pregnancy during which it is given, in other words dose and time. A large dose in rats in the early days of pregnancy results in resorption of the foetus. Large doses begun later in pregnancy produce in the female young a completely developed Wolffian duct and external genitalia of the male type; but in addition there are almost normal oviducts

and uterus, the latter however opening into the urethra between the Wolffian orifices at the site of the foetal Müllerian tubercle. The only possible explanation of these observations is that each gonoduct will develop normally if stimulated sufficiently early by a suitable concentration of its own sex hormone.

When the amount of androgen administered to the mother is slightly reduced there is no change in the male development; but the Müllerian duct now ends blindly, no longer opening into the urethra. Evidently the female hormone has been able to exercise some influence on the downward growth of the genital cords, and in the beginning of the development of the vagina. This influence was strong enough to close the primitive opening of the Müllerian duct, but not strong enough to cause the vagina to reach the perineum. It is interesting to recall that in 1904 Wood Jones[6] showed that an early change in the development of the lower genital tract in the female mammal was the closure of the opening of the Müllerian duct into the urogenital sinus, and the subsequent development of the tract below this point was entirely new. One deduction may be made from this evidence, namely, that the various tissues respond unequally to the same stimulus.

The problem with regard to the external genital organs, and urogenital sinus is quite different. Here there is a collection of organs whose primordia are neutral, which may take either male or female form depending on which sex hormone dominates them. It is frequently stated that the female external genitalia including the urogenital sinus more closely resemble the foetal form than do those of the male. This view is superficial both critically and anatomically. It is true that the lower part of the sinus and adjacent structures retain more of the embryonic form, but if one looks and dissects more deeply it is obvious at once that the upper reaches of the sinus in the male have changed little during foetal life.

Before offering any tentative suggestions to explain the male character of certain areas of this child, one must bear in mind 3 facts:

(1) There is total ignorance as to the nature and the concentration in the foetal circulation of the primitive sex hormones.

(2) It is impossible to say definitely in this case when or in what quantity the male hormone from the ovarian tumour reached the foetus.

(3) There is the unequal reaction of the various tissues of the foetus to the same quantity of the sex hormone, even if this latter were known.

It is suggested that the androgen from the ovarian tumour did not reach the foetus in effective quantity until the 3rd month of foetal life. By this time the female hormone of the foetal gonad had already produced some reactions in the accessory sex organs, probably in the upper part of the urogenital sinus. There would be the normal lag in the development of the phallus, due either to the absence of the male hormone or the presence of the female, and the urethral groove in the clitoris would be poorly developed in comparison with the corresponding groove in a penis. This poor development explains why, although the labia minora unite to advance the urethral orifice, the urethra does not canalize the clitoris. The enlargement of the clitoris cannot be called a developmental abnormality, for the same stimulus will produce enlargement of this organ in an adult, though perhaps not to the same degree.

Someone may say that this case has been reported prematurely, and that we should have waited until our knowledge about the child was complete. My answer is that one

would have to wait 3 or 4 years before more thorough investigation was attempted, and another 10 years before we could know one important fact, whether the ovaries, if there are any, would begin to function normally.

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# Basal Body Temperature Graphs as an Index to Ovulation*

BY

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IN recent years interest has been revived in the phenomenon of temperature variation in normal women during the menstrual cycle. These variations, first described more than 40 years ago, did not become significant in clinical medicine until the last two decades when the relation between ovulation, the function of the corpus luteum, and menstruation began to be properly understood. It now appears that the basal body temperature of woman is directly related to the phases of the menstrual cycle. To be explicit: the temperature is relatively low prior to ovulation, shifts to a higher level about the time of ovulation, and maintains this higher level until the next menses when it falls and the cycle is repeated. The purpose of the following presentation is, firstly, to illustrate normal temperature curves in non-pregnant and pregnant patients, and secondly, to suggest that temperature graphs constitute a simple and valuable instrument of investigation, particularly in the study of infertility.

Since the literature has recently been reviewed by the author[1] a full list of references will not be offered here. It will suffice to state that a correlation of temperature with the histology of the corpus luteum[2] and with the endometrium,[3] together with the experience of clinicians in treating infertile women gives convincing, if not conclusive, evidence that the shift in woman's basal body temperature level occurs at, or very near, the time of ovulation. In short, the "temperature-shift" in the basal temperature graph is often indicative of ovulation. (This is not the same as stating that ovulation causes the temperature-shift or that a temperature-shift necessarily accompanies ovulation.)

If the temperature-shift is to be detected, reasonable care must be employed to ensure basal conditions when the temperature is taken. All temperatures for the accompanying graphs were taken *per rectum* by the patient in the morning before rising and before taking food or liquids. "Smooth" graphs depend almost entirely upon the attention given to maintaining basal conditions. Detailed instructions may be found in one of the publications cited.[1]

The graphs shown here were selected from records aggregating about 250 months' experience accumulated by some 60 private patients all of whom recorded the *data* on special form sheets supplied by the author.† Most of the patients were eager to conceive; this desire assured the utmost co-operation. The high level of intelligence of the group also contributed to the quality of the graphs

* The substance of this report was presented (by invitation) at a meeting sponsored by the National Committee on Maternal Health in New York City, January 26th, 1945.

† A sample of these forms may be secured by a request addressed to the author at 807 Spruce Street, Philadelphia 7, Pennsylvania, U.S.A.

submitted. Although most of the patients kept graphs in order to determine the time of optimal fertility (ovulation) in the hope of conceiving, it must not be understood that they were, as a group, "sterile." Some already had children, some were fertile but required artificial insemination because of the husband's sterility, some wished to conceive in a particular month, and a considerable number wished to conceive promptly because their husbands were about to be sent overseas in military service. A few kept temperature records in order to schedule a visit to their husbands in Army camps at a time when conception would be most probable.

At this point the reader may choose to examine the graphs in detail.

Since the meat of the matter is in the graph itself, little remains but to summarize the subject by listing uses to which these basal temperature records may be put:

1. As an index of ovulation to determine:

   (*a*) The time of maximal fertility for those who wish to conceive. Charts I, II, III, IV, V, VI.

   (*b*) The "unsafe" period for those who do not use contraceptives. Charts VII, VIII.

2. As a suggestive sign of early pregnancy ("The poor man's Friedman test"). Chart IV and legend to Chart I.

3. As an aid in evaluating treatment intended to stimulate ovulation. Chart X.

4. As an aid in securing biopsies in the preovulatory (proliferative) or postovulatory (secretory) phases of the endometrium.

5. As an aid in the search for early human ova, unfertilized, fertilized, or nidated.

6. As a method of establishing a measure of "control" in studying the effectiveness of contraceptives. See legend to Chart VIII.

7. As a means of investigating some types of irregular uterine bleeding. Chart IX.

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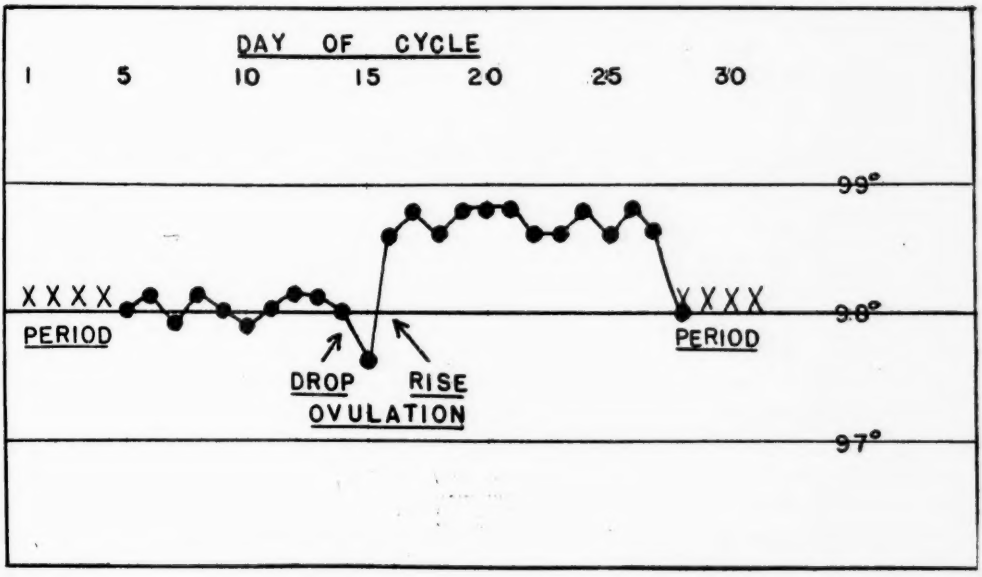


CHART I.

An idealized basal temperature graph for a hypothetical patient with a cycle of 28/4. The actual temperature is of no significance; it is the relative temperature which is important. To indicate the unimportance of the actual temperature, the degrees are noted at the right instead of at the left. The noteworthy characteristics of the normal graph are: (1) The relatively low level following menstruation, (2) the slight drop in temperature about 14 days before menstruation followed by (3), the abrupt rise in temperature. (4) The higher temperature is maintained until the next period occurs when (5) it declines sharply to the post-menstrual level. All temperatures were taken *per rectum* by the patient before she arose in the morning or took foods or liquids. Such temperatures were considered "basal." Contrary to expectations, the readings were usually in the range between 97°F. and 98°F. In the patient with a 28-day cycle, the temperature shift will be found approximately 14 days after the 1st day of menstruation. If the menstrual cycle be longer than 28 days, the shift will be found more than 14 days after the period, but it will occur approximately 14 days before the next period begins. The higher temperature level will be maintained for about 14 days. Thus far, the author has not observed a high temperature level which persisted for more than 16 days except in patients who subsequently were proved to be pregnant.

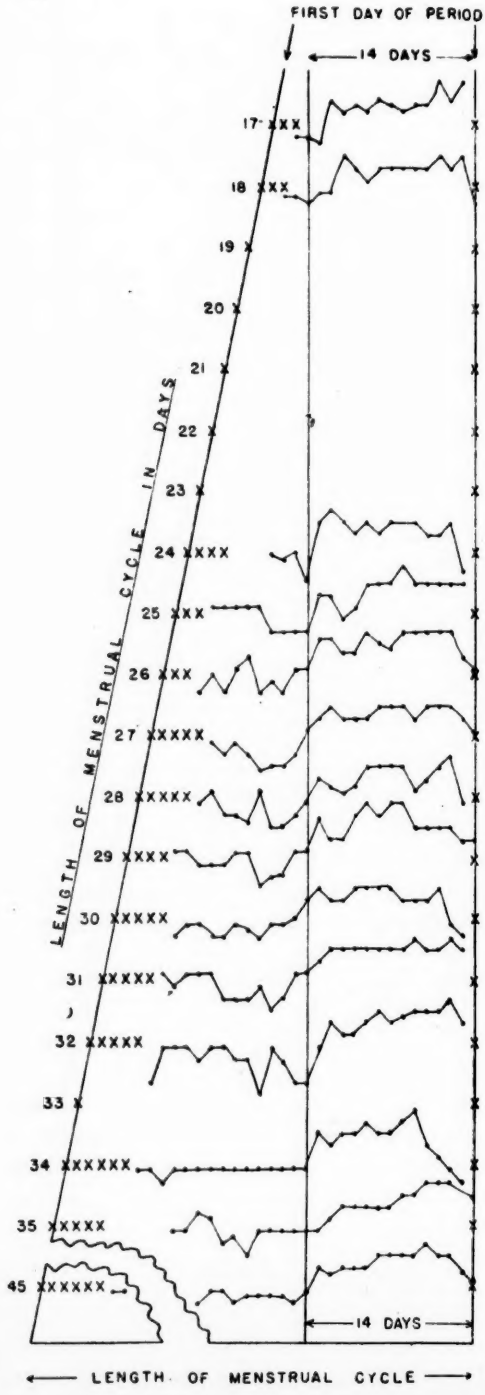


CHART II.

This chart consists of a number of temperature records actually submitted by patients. The graphs are arranged in order from the shortest cycle (17 days) to the longest cycle (45 days) observed. The 1st day of the first period is marked by an X at the left of the chart and the 1st day of the succeeding period by an X at the right of the chart. It will be noted that, regardless of the length of the cycle, the temperature shift occurs approximately 14 days before menstruation. The graphs for the 34- and the 45-day cycle constitute the only "control" which the author has been able to secure. These temperatures were recorded by the author's wife under as nearly true basal condition as one is likely to obtain in the home. Taken as a whole, this exhibit of graphs constitutes a satisfactory demonstration of the current belief that ovulation precedes menstruation by approximately 14 days, no matter what the length of the menstrual cycle.

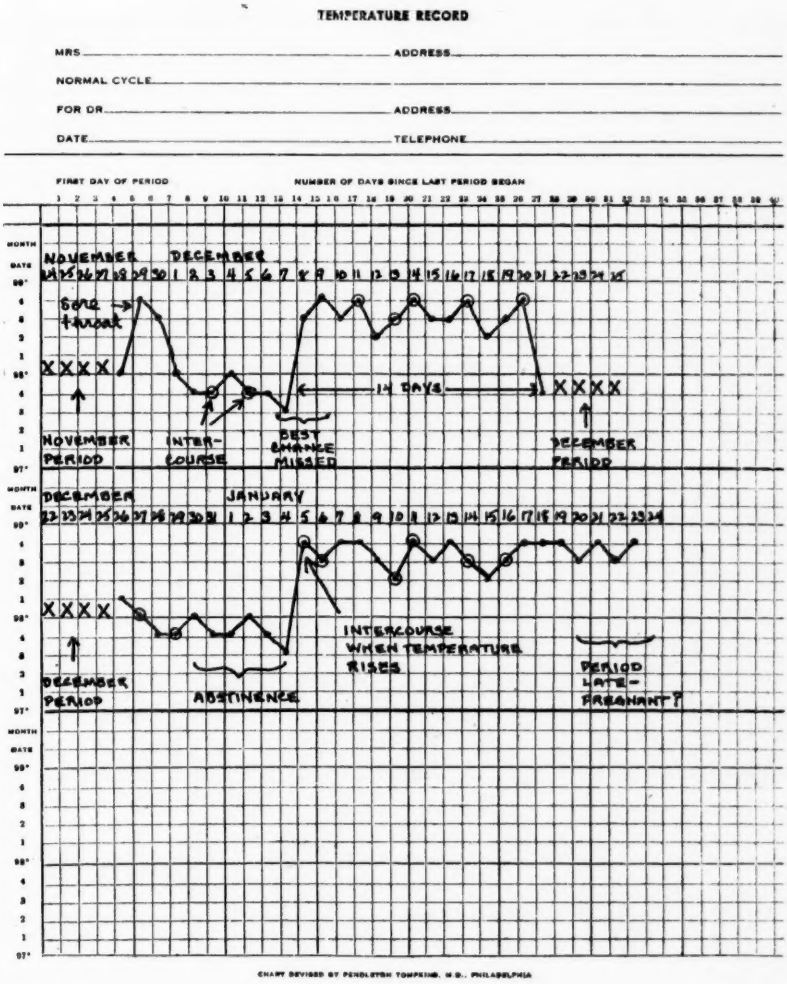


CHART DEVISED BY PENDLETON TOMPKINS, M.D., PHILADELPHIA

CHART III.

A reproduction of the sample graph supplied to patients who are using temperature records as an aid in determining the optimal time for conception. The patient is instructed to make a notation of any indisposition, such as a sore throat, which might account for fever. Intercourse is noted by a circle around the temperature. The first graph shows that, although intercourse was frequent, there was no chance of conception because coitus did not occur at the time of the temperature shift (ovulation). The second graph illustrates the value of the first graph in establishing the "pattern" peculiar to the individual patient. If the patient's menstrual cycles are equal in length, the temperature shift will fall at approximately the same point each month. In the second graph, a period of abstinence preceded the expected temperature shift and, as soon as the shift occurred, intercourse took place. As a result, in this hypothetical case, the patient conceived.

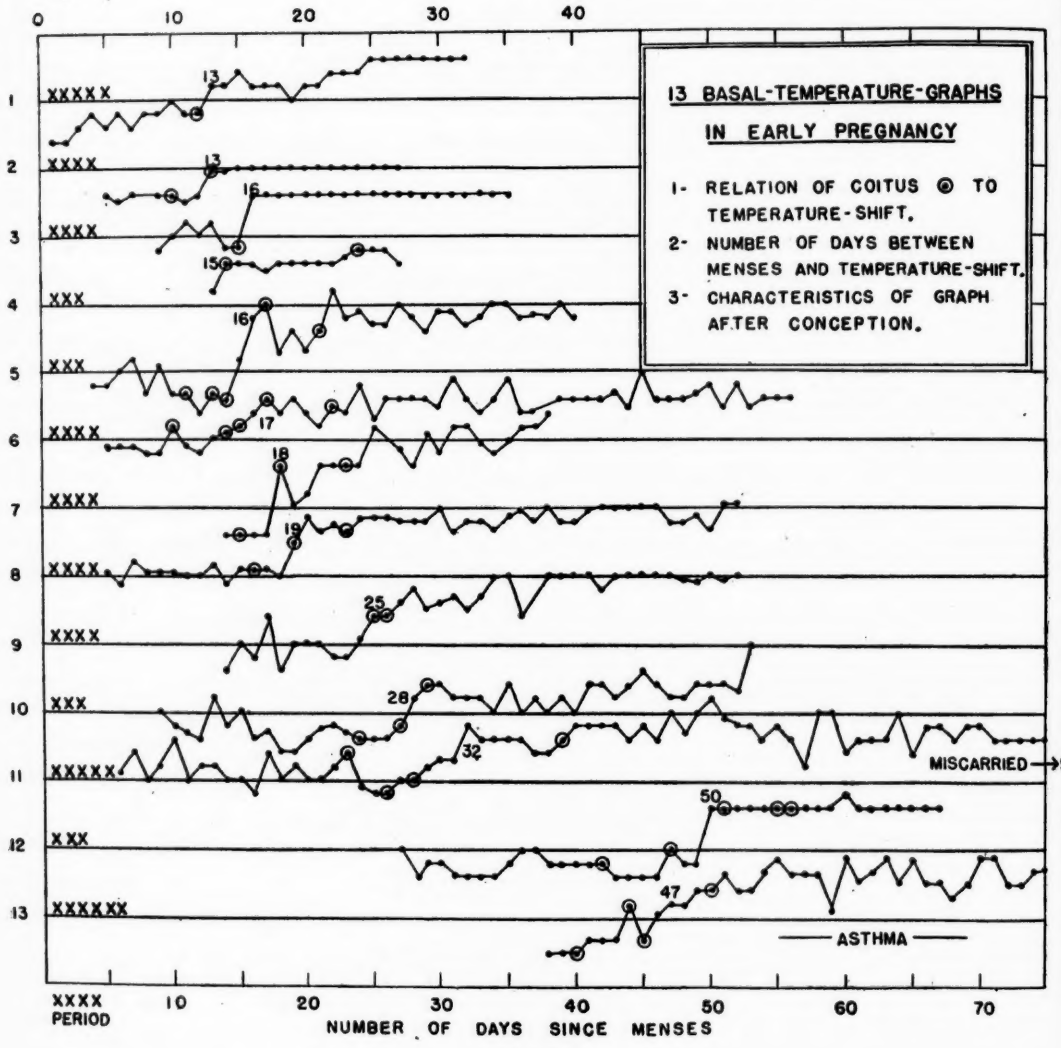


CHART IV.

This exhibit demonstrates the characteristic temperature curve after conception. It will be noted that the high post-ovulation temperature level is maintained for some weeks. In most cases the temperature is strikingly level after conception. It should also be noted that, in every case, intercourse occurred very close to the date of the temperature shift. Some of the graphs deserve special mention: Graph 1 represents artificial insemination performed on the 12th day after menstruation because the temperature graph of the previous month indicated that the temperature shift would occur on the 13th or 14th day. The patient's husband is irremediably sterile. She now has two healthy children as a result of insemination on two occasions. Conception following infrequent intercourse is shown in Graph 2 (twice in the month), Graph 3 (once in the month), and Graph 4 (twice in the month). The graphs in the lower half of the chart were drawn by patients with long or irregular menstrual cycles. The patient who drew Graph 11 miscarried 93 days after her last period. Attention is called to the very irregular temperature recorded after conception. Whether this irregular temperature is typical in cases of miscarriage or whether it is of prognostic value remains to be learned. The author has not observed another such case. The temperature in Graph 13 is also irregular, but the patient suffered from asthma at the time. The diagnosis of pregnancy in Case 11 was confirmed both by a Friedman test and by examination of the products of miscarriage. All the other patients have either delivered or are at present very close to term, so that there is no question of pregnancy. From this exhibit it will be seen that the temperature shift is a useful index, no matter how long it occurs after the period.

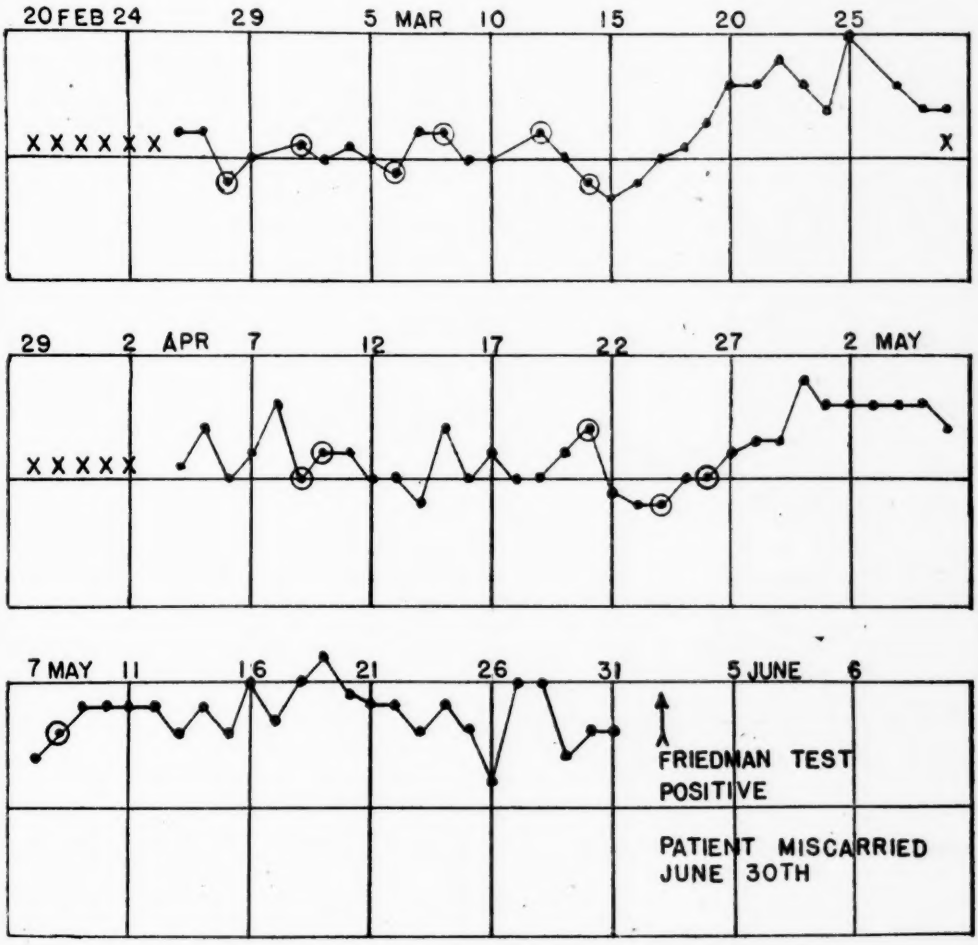


CHART V.

Graph 11 in Chart IV is here shown in more detail together with the graph of the preceding month. Again, the value of the graphic pattern is manifest. Conception did not occur in March, probably because intercourse occurred too early in the month. However, in April, the timing was better and conception followed. There has been considerable speculation as to whether in the human, as in the rabbit, sexual stimulation causes ovulation. The author has not found, in this graph or in others, any conclusive evidence that such is the case. There are many examples, such as the above, in which ovulation (the temperature shift) occurred shortly after intercourse, but, in every such instance, the date of temperature rise was anticipated on the basis of the record of the previous cycle.

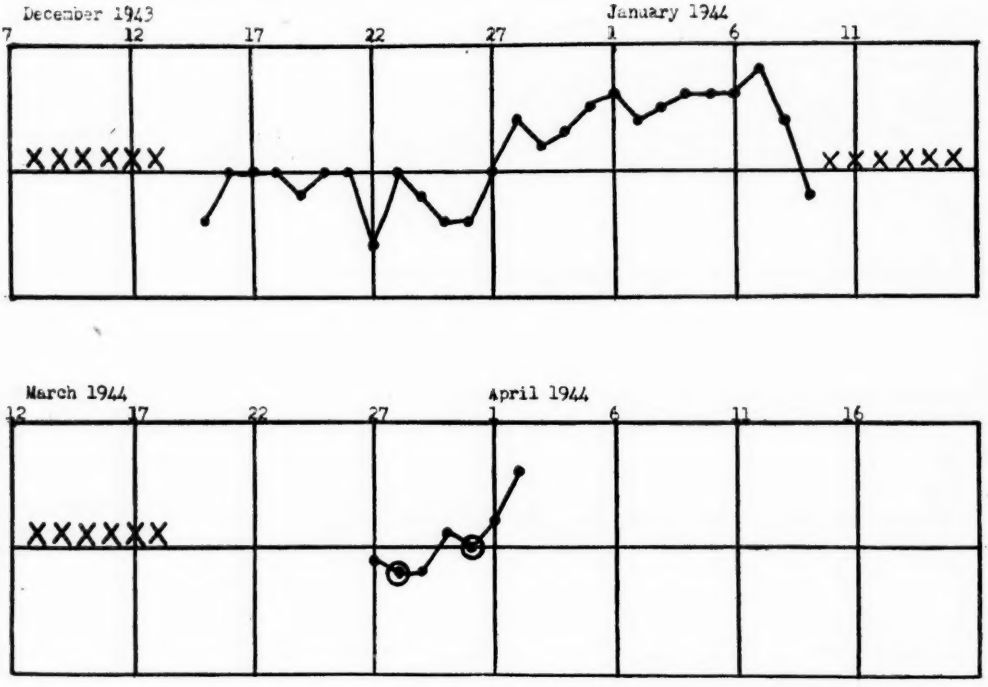


CHART VI.

This chart exemplifies the common practice of keeping a record for only a few days each month at the time when ovulation might be expected. This patient conceived on March 31st (probably not on March 28th) and was delivered on December 12th, 1944, after induction of labour.

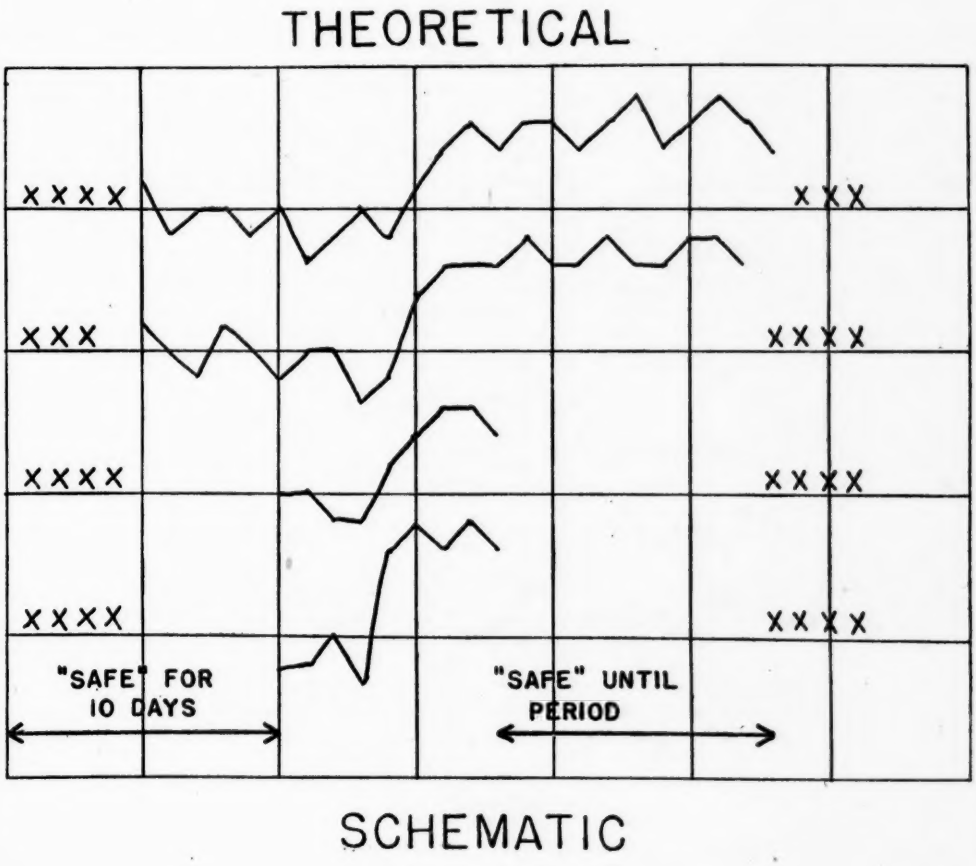


CHART VII.

This chart illustrates the use of temperature records as a means of birth control for patients who do not use contraceptives. The patient is instructed to keep a complete record for 2 months; thereafter she begins the temperature graph shortly after menstruation has ended and keeps the record for 3 or 4 days after the temperature shift has occurred. Probably there is very little chance of conception after the temperature shift, but, obviously, the days before the temperature shift are much less certainly safe. This method of detecting the "safe" and "unsafe" periods entails more bother than the method of Ogino-Knaus, but, in the case of a patient with very irregular periods, may be more reliable.

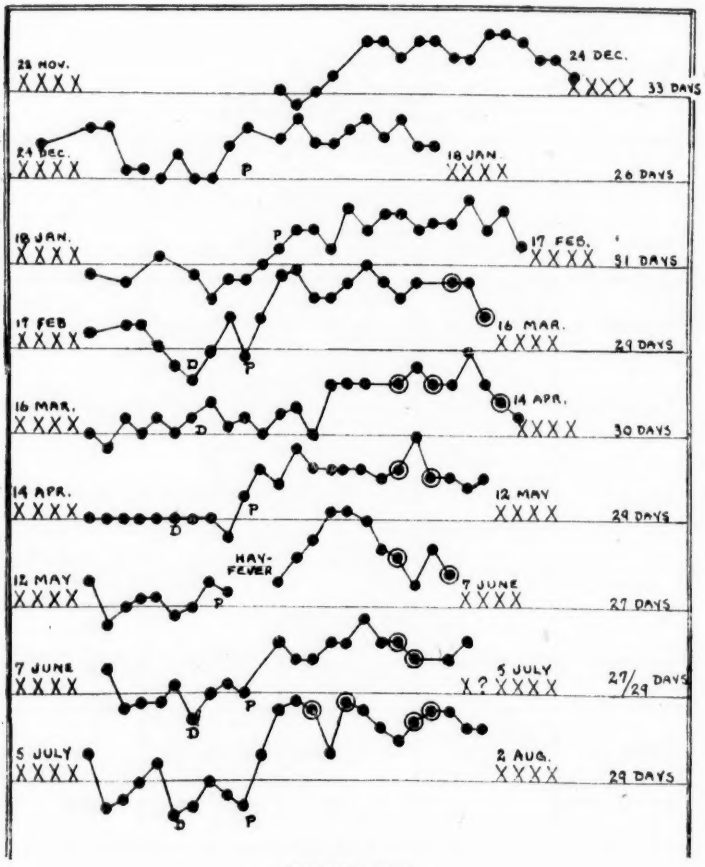


CHART VIII.

A record of basal body temperatures for 1 year (13 consecutive menstrual cycles). The patient was 28 years of age when this chart was commenced and had just had her second baby. In fact the baby was born on October 2nd, and the period of November 22nd was the first postpartum menstruation. The graph was kept in order to determine the time of ovulation so that another pregnancy might be avoided. Previously the patient had employed the "rhythm system" of birth control as commonly taught, without success. Accordingly she had no very great faith in the temperature graph and it will be noted that only on one occasion is it recorded that intercourse occurred prior to the temperature shift. This woman is a keen observer and has noted the date of abdominal pain, probably *mittelschmerz*, by the letter P, and by the letter D indicated a mucus discharge which occurred with regularity about 4 days before the temperature shift. Incidentally, if this patient had used any contraceptive device or chemical during the year illustrated it is probable that the contraceptive would have received credit for protecting her from pregnancy. As the graph shows, she actually had no opportunity to conceive, since intercourse never occurred near the time of ovulation. Perhaps a controlled study of the effectiveness of a contraceptive would show that it protected a fertile individual from conception not for, let us say, 50 months, but that it protected her from conception when intercourse occurred at the time of ovulation on 50 occasions.

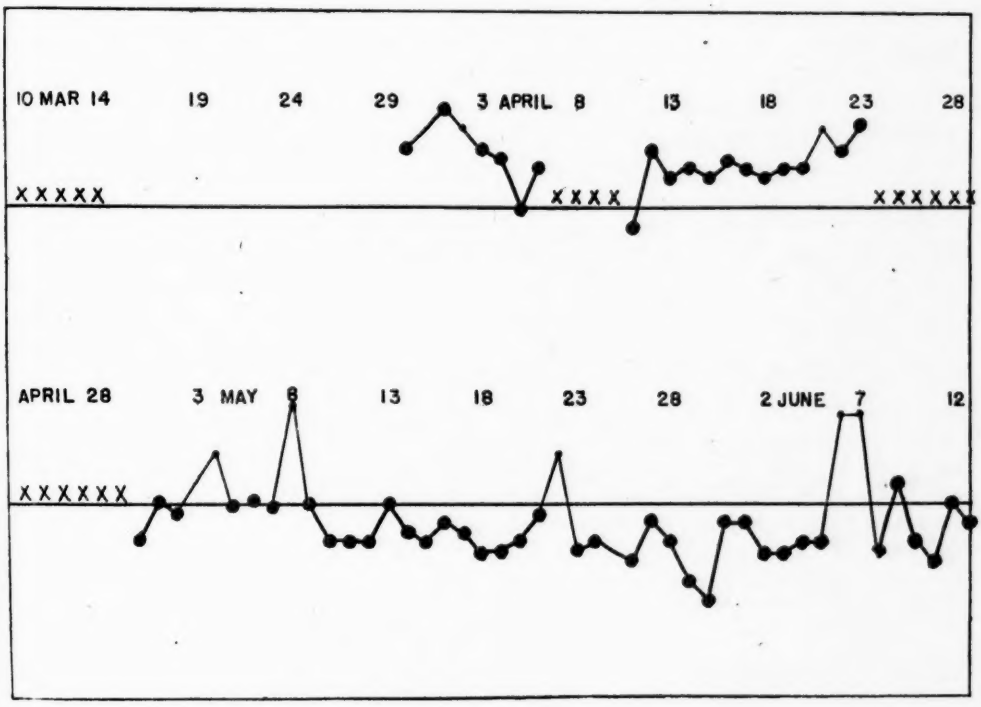


CHART IX.

This graph was kept by a patient of 47 whose menstrual irregularity caused considerable social inconvenience. The chart was begun on March 30th simply as a means of predetermining the date of menstruation. As it turned out, vaginal bleeding occurred only twice subsequently, so this graph represents the last menstruation and the beginning of the menopause. It is interesting to note that, in the menstrual cycle of April 7th to 24th, the temperature shift occurred immediately after the end of the period. Subsequently, the temperature remained elevated for 12 days. The temperatures indicated by the small dots and fine connecting lines are temperatures taken after several alcoholic drinks the preceding evening. These temperatures may be disregarded in reading the graph.

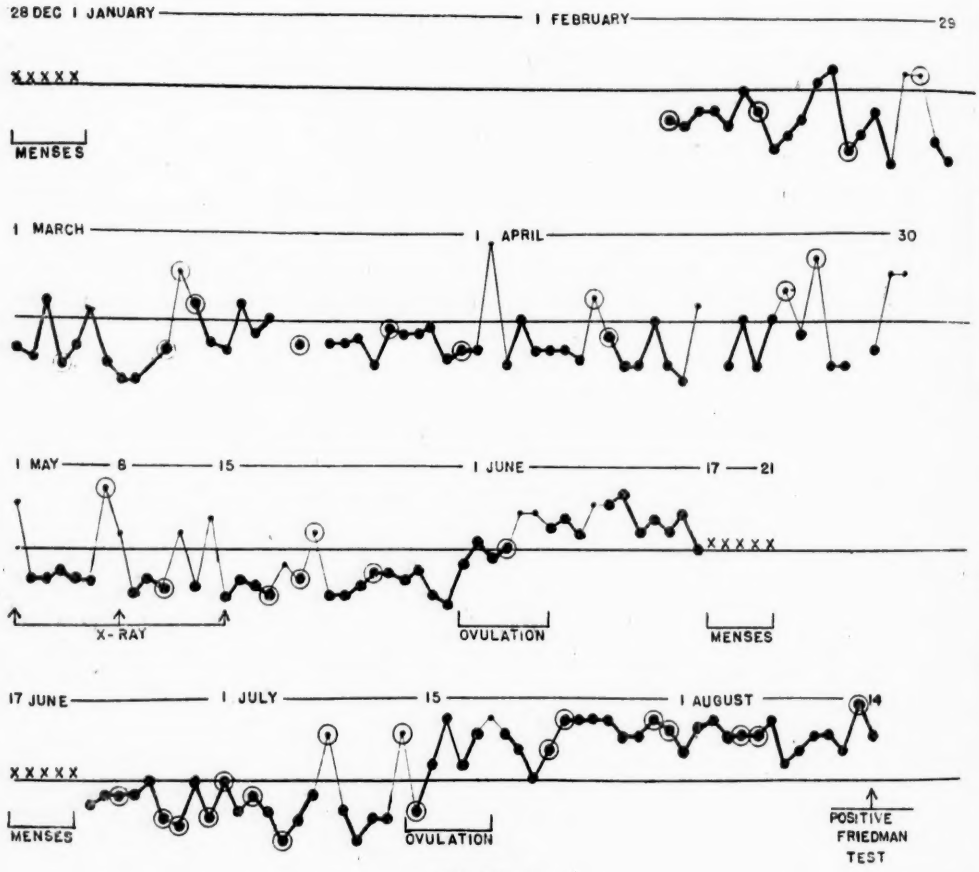


CHART X.

This graph was kept by a patient of 30 years whose periods occurred " 2 or 3 times a year." The temperature graph was begun on February 10th, 6 weeks after the last menstruation. The small dots and fine lines indicate temperatures taken after alcoholic drinks and may be disregarded in reading the graph. There was no evidence of ovulation prior to May 1st. On that date the patient became urgently interested in conceiving because she thought her husband was about to go overseas. For this reason, and without the usual preliminary endocrinological study, X-ray treatments were administered after the technique of Mazer and Baer.[4] Three treatments were administered at weekly intervals and, about 2 weeks after the last treatment there was evidence of ovulation. As shown above, menstruation occurred on June 17th, the 1st period since the preceding December. Following the June period, there was evidence of ovulation about July 15th. Subsequently, the temperature remained high and, on August 14th, the Friedman test was positive. At the present writing the patient is 6 months pregnant and is in good health.

# Changes in Numbers of Circulating Blood Platelets following Experimental Traumata

BY

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## INTRODUCTION.

ALTHOUGH it has long been recognized that there is an increase in the numbers of circulating platelets in the postoperative period, very little is known at present concerning the factors which bring about this change. During the 2nd week after operation, when the platelets usually reach their maximum, it is found that thrombosis occurs more frequently than at other times (Hueck,[1] Dawbarn *et al*,[2] Kristensen,[3] Øwre,[4] Willinsky,[5] Nicolaysen,[6] Brock,[7] Pilcher,[8, 9] Barker *et al*,[10] Wright[11]). It has been estimated that pulmonary embolism follows about 0.77 per cent of all major operations (Barker *et al*.[12]), while from autopsies it is known that thrombosis of some degree, clinically suspected or not, occurs in a far greater proportion of patients. Burke[13] showed by careful dissection at postmortem examinations that thrombosis at some site was present in 24 per cent of all cases, both medical and surgical, and he quotes the following figures for similar observations abroad: Germany (Bela[14]) 14 per cent, Australia (Cleland[15]) 6.3 per cent, and Switzerland (Wantoch[16]) 7.7 per cent. In this country Pilcher[9] reported 261 cases of thrombosis and pulmonary embolism diagnosed clinically during 10 years at University College Hospital, London, among which the mortality-rate was 16.8 per cent, 4.6 per cent being due to fatal pulmonary embolism following an earlier clinical diagnosis of thrombosis elsewhere.

The problem of postoperative thrombosis is, therefore, one which merits considerable study, and the present series of experiments have been devised in order to throw light upon some of the factors influencing the rise in blood platelets in the critical period. A systematic experimental study of postoperative platelet changes has consequently been undertaken by subdividing the component parts of major operations into stages in order that the effects of various constituent procedures may be studied separately.

## METHODS.

Platelet and red cell counts were carried out upon blood obtained by razor cuts in the marginal ear veins of rabbits. Only freely flowing blood, dropping about once a second, was used. The blood was diluted with Rees-Ecker diluting fluid, modified by the addition of 2 per cent (instead of 0.2 per cent) formalin, in order to ensure the immediate fixation of the platelets. In order to prevent any platelets adhering to the dry stem of the pipettes, diluting fluid was first drawn up to the 0.5 mark and this was followed by the same quantity of blood. Platelet and red-cell counts were both made from the same sample, by the direct wet method using a Bürcker counting chamber.

Veterinary nembutal, 1 c.cm. (60 mg.)

per kilo body weight, injected intraperitoneally, was used as anaesthetic in every instance.

### Operative Procedure.

Six full-grown rabbits, fed upon fresh vegetables, oats and bran, were used for this series of experiments. The interval between each operative stage in any animal was at least 5 weeks. Observations were made upon the numbers of platelets and red cells before each operation, immediately after (within 1 hour) and on the 4th, 7th, 10th, 14th and 21st postoperative days.

The graded operations were as follows:

1. *Bleeding with minimal surgical trauma.* The marginal ear vein was cut transversely with a sharp razor, care being taken not to damage the surrounding tissues. Each animal was bled 35 to 40 c.cm. (estimated to be about 25 to 30 per cent of blood volume) in 10 to 15 minutes.

2. *Simple skin incision.* Under anaesthesia, a longitudinal incision 2 to 4 cm. long was made through the skin and superficial fascia in the left paravertebral region about 1½ cm. from the midline. The repair sutures were catgut.

3. *Skin and muscle incision.* A similar incision was made in the right paravertebral region and was extended deeply to include the skin, fascia, fibrous muscle sheath and muscle tissue to a depth of about 3 cm. Very little bleeding occurred and the tissues were repaired in three layers Operation time was about 15 minutes.

4. *Non-surgical destruction of muscle tissue.* 2 c.cm. of a 2.0 per cent suspension of Neosyl (aluminium silicate) in normal saline was injected into the thigh muscles upon the left side, about 2 cm. below the head of the femur.

5. *Coeliotomy with manipulation of the gut.* A midline abdominal incision, passing downwards for 3 or 4 cm. from the xiphisternum was made to expose the abdominal viscera. Loops of intestine were withdrawn from the abdominal cavity on to warm cloths and for 3 minutes were gently handled in a manner similar to that used when inspecting the gut systematically for a lesion in man. Care was taken not to pull upon the mesentery. The intestinal loops were replaced and the incision repaired in layers.

6. *Coeliotomy with autolysis of muscle strip.* A midline incision about 1½ cm. in length, was made in the middle abdomen. The linea alba was opened for about 1 cm. and from the sides of this incision strips of muscle, the whole thickness of the abdominal wall, were removed. These strips were crescentic, 1 cm. long, and about ½ cm. wide, in their central portions. They were introduced deeply between the folds of gut, care being taken to disturb the latter as little as possible. The repair of the abdominal wall was performed as before.

7. *Non-surgical sterile irritation of the peritoneum.* A low-grade peritonitis was produced by intraperitoneal injection of 5 c.cm. of 2.0 per cent suspension of Neosyl in normal saline.

8. *Splenectomy.* The figures for this stage were obtained upon a different group of 6 rabbits as it was considered undesirable to use animals after the production of peritonitis since the surgical technique would be complicated by the presence of adhesions. An incision similar to that used in Stage 2 was made. The exposed stomach was drawn forward and the spleen removed after the successive ligation of the splenic vessels without previous clamping (see Steiner and Gunn[17]). Repair was carried out as before.

### Results.

The average red-cell and platelet counts for the initial observations and for the stated postoperative days are set out in Table I.

Fig. 1 shows the average percentage change from the initial counts at various intervals after each traumatic procedure.

The immediate effect of haemorrhage (Stage 1) was to reduce the red-cell and platelet counts proportionately. Both counts returned to within normal limits by the 14th day, the restoration of the platelets being rather more rapid than that of the red cells.

The general configurations of the curves in each of the other 7 operative procedures were similar, though the extent of the response varied. In all the stages the red-cell counts remained steady throughout. The platelets, however, in all instances showed an increase in numbers, reaching a maximum in 7 to 10 days and thereafter returning to normal by the 21st day. The extent of the rise appeared to bear a direct relation to the severity of the operation. It was hardly noticeable in Stage 2 when the skin alone was damaged (10 per cent rise) and became most marked in Stages 5 and 8 when there was both muscle damage and peritoneal irritation with tissue destruction (80 and 123 per cent). In Stages 3, 4, 6 and 7, where the degree of tissue damage may be assumed to be roughly the same, the increase over the initial count in the number of circulating platelets was remarkably similar, being 50, 48, 62 and 62 per cent respectively.

TABLE I.

| Day | | Initial | Within 1 hour | 4th day | 7th day | 10th day | 14th day | 21st day |
|---|---|---|---|---|---|---|---|---|
| Stage 1 ... ... | R.B.C.s | 6.10 | 4.52 | 4.85 | 5.14 | 5.55 | 5.77 | 6.00 |
| Haemorrhage ... | Platelets | 549 | 350 | 420 | 512 | 500 | 529 | 546 |
| Stage 2 ... ... | R.B.C.s | 5.81 | 5.79 | 5.81 | 5.38 | 5.75 | 5.81 | 5.78 |
| Skin incision ... | Platelets | 554 | 548 | 570 | 609 | 593 | 575 | 538 |
| Stage 3 ... ... | R.B.C.s | 6.02 | 5.95 | 5.88 | 5.90 | 6.09 | 6.07 | 6.00 |
| Muscle incision ... | Platelets | 575 | 562 | 610 | 770 | 852 | 739 | 584 |
| Stage 4 ... ... | R.B.C.s | 5.91 | 5.92 | 5.95 | 5.87 | 5.80 | 5.83 | 5.79 |
| Muscle destruction | Platelets | 544 | 545 | 601 | 742 | 801 | 694 | 569 |
| Stage 5 ... ... | R.B.C.s | 5.97 | 5.85 | 5.84 | 5.83 | 5.93 | 5.93 | 5.97 |
| Manipulation of gut | Platelets | 593 | 580 | 645 | 822 | 1027 | 779 | 600 |
| Stage 6 ... ... | R.B.C.s | 5.98 | 5.94 | 5.94 | 5.97 | 5.92 | 5.92 | 5.83 |
| Autolysis of muscle strip | Platelets | 514 | 508 | 598 | 714 | 830 | 654 | 526 |
| Stage 7 ... ... | R.B.C.s | 5.79 | 5.94 | 5.78 | 5.77 | 5.78 | 5.80 | 5.79 |
| Sterile peritonitis | Platelets | 554 | 554 | 572 | 682 | 770 | 677 | 553 |
| Stage 8 ... ... | R.B.C.s | — | — | — | — | — | — | — |
| Splenectomy ... | Platelets | 541 | 541 | 805 | 1162 | 1120 | 815 | 641 |

Average red-cell and platelet counts on the various days.
R.B.C.s in millions. Platelets in thousands.

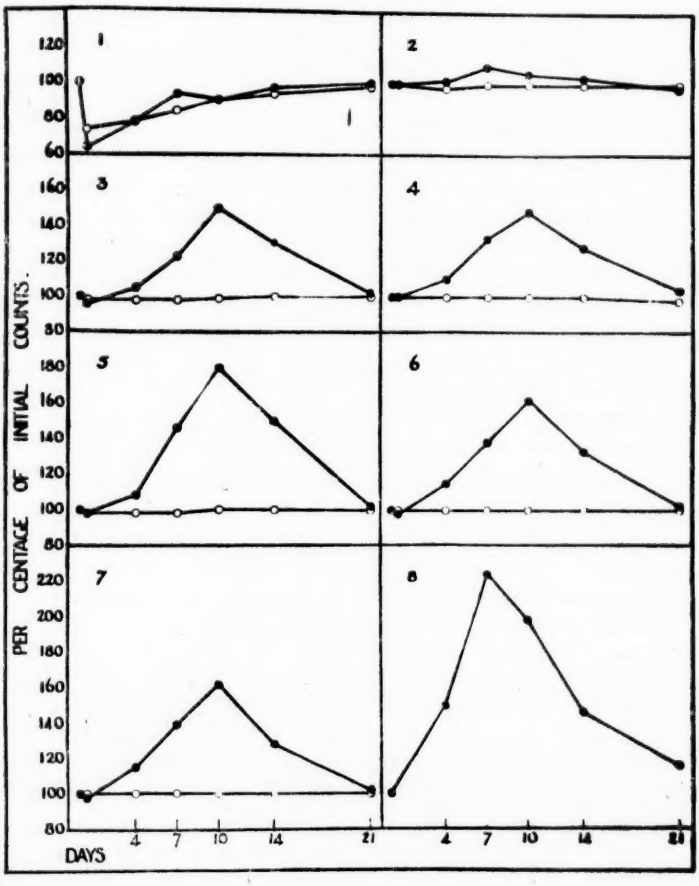
## DISCUSSION.

From a search of the literature upon the blood platelets changes following surgical procedures, it is surprising how piecemeal is the present state of knowledge. The wide variety of methods used for the actual

counting, together with differences of types of subjects or species of experimental animal, renders it difficult to compare the findings of the many investigations. From such observations as have been made, however, it is now generally believed that tissue damage is followed a week or 10 days later by a platelet response roughly proportional to the extent of the area traumatized, but the nature of the chemical stimulus concerned has not been elucidated. The present study, while repeating some of the observations made by previous workers, has attempted to systematize the field of investigation both by using a standard method of platelet counting throughout and by carrying out a graded series of surgical procedures upon the same group of animals.

FIG. 1.

Average numbers of platelets (solid circles) and red cells (hollow circles), expressed as percentages of the initial counts on the various days following operation. Curves numbered (1–8) to correspond with the experimental stages described in the text.




In 1928 Dawbarn, Earlam and Evans[2]

published their study upon platelet counts in man following operations and parturition. Their observations, now confirmed both clinically and experimentally by other workers, showed that haemorrhage alone was not followed by a rise in the numbers of circulating platelets, but that a rise occurred after surgical procedures. After considering several possible causes of platelet stimulation, they concluded that the only feature common to all their cases which showed the increase in platelet count, was tissue damage with the consequent absorption of autolytic products. Further, they showed that to some extent the degree of response was correlated with the extent of damage. Recently Adams[18] has published a study upon a group of 51 miscellaneous surgical cases in man which showed percentage platelet elevations of a similar order to those found by Dawbarn and his associates. Adams noted that while small responses are evoked by minor operations and large ones by procedures involving much dissection and manipulation, the great majority of operations, whatever their nature, caused a remarkably constant increase in platelet numbers.

These observations, both for haemorrhage and for operations, are confirmed by the present experimental study. Haemorrhage was followed by an immediate and equal fall in both red cells and platelets. The subsequent rise in both took place steadily during the ensuing 3 weeks, but the count for the platelets never rose above that initially observed. The other procedures, viz., muscle incision, chemical destruction of muscle, autolysis of muscle strips and sterile peritonitis showed practically identical platelet responses, the increase in count being to 150 to 160 per cent of the initial figures. This percentage increase is very similar to those found in man by Dawbarn *et al.* and by Adams.

Other workers have reported elevated platelet counts in circumstances in which there has been no surgical intervention, but in which absorption of the products of tissue autolysis has occurred. Brock and Rake[19] found an increase in platelets in tubercular patients with pleural effusions, and they also showed that tuberculous rabbits when treated with tuberculin showed a similar rise. Steiner and Gunn[20] found a rise in platelets in rabbits following exposure to ultra-violet light and, from a comparison of these findings with those of the postoperative period, they concluded that both were due to absorption of breakdown products of injured tissues. Pohle[21] made the same suggestion to explain the increased platelet count following menstruation in women. Fleming,[22] using dogs and rats, showed that a small platelet rise of 10 to 15 per cent followed irritation of the lungs with nitrous fumes. The numbers increased in the circulation within an hour, but unfortunately observations were not made on days subsequent to the exposure. Zschau[23] showed that intravenous injection of protein solutions (casein) into rabbits was followed by a definite rise in the numbers of circulating platelets, which began about the 8th day and returned to normal in 2 to 3 weeks. Under sterile conditions he also excised strips of lumbar muscle which he then replaced in the sites of excision to undergo autolysis. The platelet increase following this procedure was very similar both in duration and extent to that following autolysis of muscle strip in the abdominal cavity in the present series. König[24] found that a number of proteins or protein breakdown products (casein, novoprotein, lucarnol, muscle press juice, xanthine, guanosine and adenylic acid) caused a rise in the platelet count. He concluded that it was the autolysis of nuclear material which was the chief factor in stimulating the platelet response in the postoperative period.

That the rise in platelets in the circulation is due to tissue damage is supported by the present study. The increase in their numbers following destruction of muscle with Neosyl (Stage 4) is closely comparable with that after muscle incision (Stage 3). Similarly a comparable increase occurs with sterile neosyl peritonitis (Stage 7) and with autolysis of muscle in the abdominal cavity (Stage 6). In both these latter conditions tissue breakdown products entered the blood-stream from the peritoneum. The most marked increases were observed in Stages 5 (manipulation of gut) and 8 (splenectomy) and may well have been due to the considerable tissue injury intentionally inflicted. The large rise following Stage 5 of the present series falls into line with the observations made by Dawbarn and his colleagues and by Adams, that the greater the manipulation of the gut the higher the subsequent platelet count. Splenectomy, also, is known to cause a very marked platelet response both in man (Brock and Rake[19]) and in animals (Liles,[25] Steiner and Gunn,[17] König,[24] Wright[26]). This marked rise may be due partly to the operative trauma caused by considerable manipulation of the viscera, and partly to the extirpation of the organ which is believed to remove old or redundant platelets from the circulation.

It would seem, therefore, that these graded animal experiments support the hypothesis that tissue damage resulting in autolysis is an important factor in initiating the increase in platelet numbers in the postoperative period, and that additional light may well be thrown upon the nature of the ultimate stimulus by the further investigation of tissue extracts and their chemical components.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# A Case of Retroperitoneal Haemorrhage Causing Death in Late Pregnancy*

BY

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A MARRIED woman, 30 years old, attended our antenatal clinic in the 28th week of her 2nd pregnancy. During an illegitimate pregnancy 4 years ago she had been treated for gonorrhoea and had borne normally a full-time healthy child. Evidence of venereal disease was not found now; she was very well and was sent to a clinic nearer her home for regular supervision until later pregnancy. Returning here at the 36th week for routine inspection she was found to have oedema of the legs and the blood-pressure was 160/100 mm. Hg. She was urged to stay in hospital but preferred to arrange her domestic affairs before admission. However, on the morning following her visit to the clinic she was brought in unconscious, ashen and pulseless and died within half-an-hour. At the moment of death Caesarean section was performed and a new-dead, mature child was delivered. The laparotomy revealed a little dark fluid in the peritoneal cavity and a massive retroperitoneal haemorrhage on the left side. The uterus was not ruptured nor was there any evidence of other visceral injury or external violence. The bladder contained a few drops of clear urine.

The husband said that sexual intercourse had taken place during the night and twice that morning. Following this second act the wife described sharp pain in the left side, became faint and looked ghastly. He gave her tea in bed and, as she became worse, got an ambulance and brought her to hospital. She was dead in 2 hours from the beginning of the illness.

*Postmortem examination* (6 hours later).

The abdomen contained a great retroperitoneal blood-clot, chiefly on the left, extending from the diaphragm to the brim of the pelvis and across the midline halfway towards the ascending colon. (Fig. 1). The descending colon, pancreas and origin of mesentery of the small intestine were all pushed forward. There were, roughly, 2 litres of blood in the retroperitoneal tissues, a good proportion of it lying around the left kidney. The left broad ligament was expanded by clot to a thickness of 2 cm., but there was very little blood in the recto-vaginal septum or in the right broad ligament and none in the inferior pelvic fascia. Aneurysm or rupture was not found on dissection of the abdominal and pelvic vessels. The heart (380 g.) showed a slight left ventricular hypertrophy and scattered subendocardial haemorrhages. The aorta and its branches were elastic, smooth-

* Shown at meeting of Section of Obstetrics and Gynaecology, Royal Society of Medicine, March 16th, 1945.

walled and practically free from atheroma. The inferior vena cava was patent, but together with its tributaries had been compressed by the retroperitoneal blood-clot. The tonsils and tonsillar lymph-nodes were enlarged. There were scattered superficial erosions of the gastric mucosa. The liver (1,320 g.) was soft, friable and pale brown, its substance shrunken under a wrinkled capsule. The dorsal surface of the right lobe showed a few scattered subcapsular haemorrhages. The kidneys were normal in size and remarkably pale. Their capsules stripped easily, revealing a smooth surface. The cut surface showed a dead-white cortex of normal thickness fairly well differentiated from the medulla. The retroperitoneal blood-clot had extended along both renal arteries into the connective tissue of the pelves. Apart from the gestational hypertrophy of the pale uterus the pelvic organs appeared normal. The spleen (110 g.) was pale and flabby. The pituitary (1.2 g.) showed gestational enlargement. The brain (1,220 g.), respiratory system, thyroid (50 g.), adrenals, pancreas and vertebral bone-marrow were all free of gross lesion.

*Microscopic findings*:

*Liver*. Numerous sections of the subcapsular haemorrhages show only dilatation and engorgement of the peripheral portion of the sinusoids of some of the lobules. In one area there is a more advanced lesion. The capillaries of the portal tract are distended and the surrounding connective tissue is infiltrated with free red cells, lymphocytes and occasional polymorphonuclears (Fig. 2). The junction of the peripheral sinusoids with the portal tracts is marked in a few places by the deposition of clumps of fibrin (Fig. 3). The cytoplasm of the neighbouring hepatic cells has been compressed, the nuclei are swollen and show an occasional mitosis (Fig. 4). *Kidney*. The capillaries of the glomerular tufts show a moderate fibrillary oedematous swelling of their basement-membranes (Fig. 5). A few glomeruli show a more advanced ischaemic sclerosis and there is an early elastic hyperplasia of occasional interlobular arterioles. The afferent arterioles are normal. *Cardiac muscle*. The subendocardial muscle-fibres are swollen, vaculated and degenerate and are separated by free interstitial collections of red cells (Fig. 6). *Retroperitoneal clot*. The fat is replaced by masses of red cells intersected by festoons of agglomerated platelets. The capillaries appear normal. *Stomach*. The superficial mucosal erosions are infiltrated with polymorphonuclears. Sections of the other organs do not show any striking changes except perhaps for an unusually marked development of the theca-luteal cells in the ovary.

## Discussion.

The pre-eclamptic clinical signs of hypertension and oedema were thus found to be based upon pathological changes in the liver, heart and kidneys typical of eclampsia.

In theory, the source of the retroperitoneal haemorrhage may have been arterial, venous or capillary. We may easily have missed a miliary congenital aneurysm (a source of spontaneous intra-abdominal haemorrhage suggested by Bruce[1]), but as far as we know, such aneurysm has not as yet figured in the literature. Rupture of uterine veins in pregnancy has been described; in these cases there is gross intraperitoneal haemorrhage.[2] We may have overlooked a ruptured pelvic vein in this case, but the failure of the blood to track down into the inferior pelvic fascia or into the rectovaginal septum forbids such a source. A spontaneous perirenal gross capillary oozing has been described in

Fig. 1.

Retroperitoneal blood-clot removed at postmortem together with the kidneys, aorta, inferior vena cava and the pelvic organs. The main mass of clot can be seen around and below the left kidney.

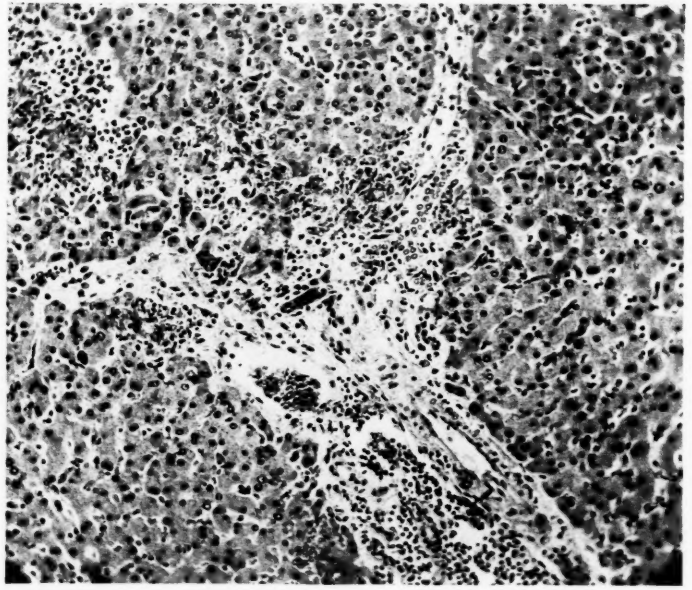

FIG. 2.

Liver. (Microphotograph × 112, Formol-Zenker, Haematoxylin and Eosin.) The portal-tract-capillaries are dilated and the connective-tissue infiltrated with red cells and leucocytes.

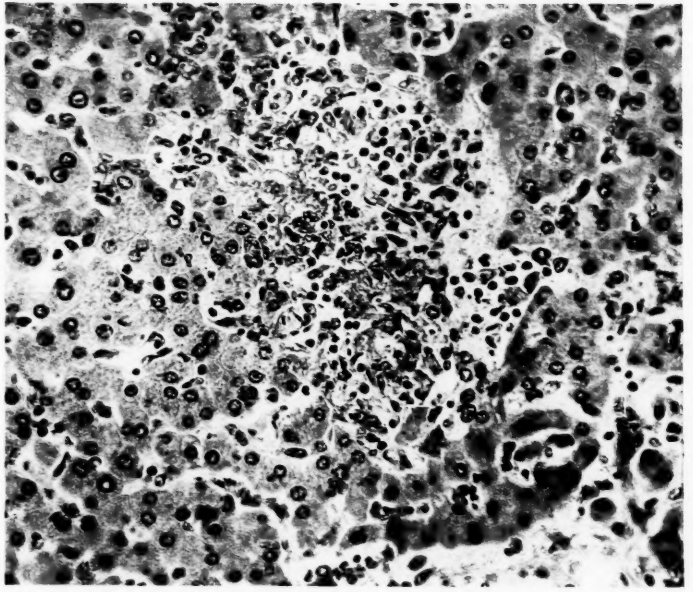

FIG. 3.

Liver. (Microphotograph × 240, Formol-Zenker, Haematoxylin and Eosin.) Fibrin-deposits are seen at the centre of the field, at the junction of peripheral sinusoids with the portal-tracts.

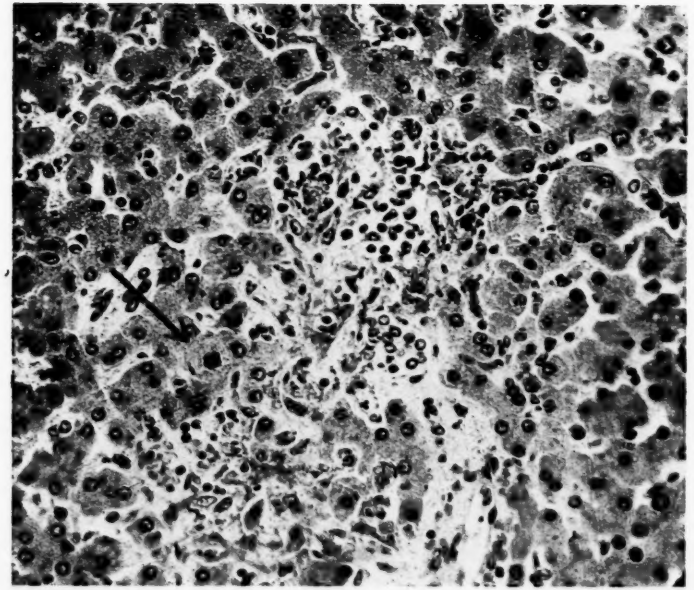

FIG. 4.

Liver. (Microphotograph × 240, Formol-Zenker, Haematoxylin and Eosin.) This shows disorganisation of hepatic cells at the periphery of a lobule and a mitosis marked by an arrow.

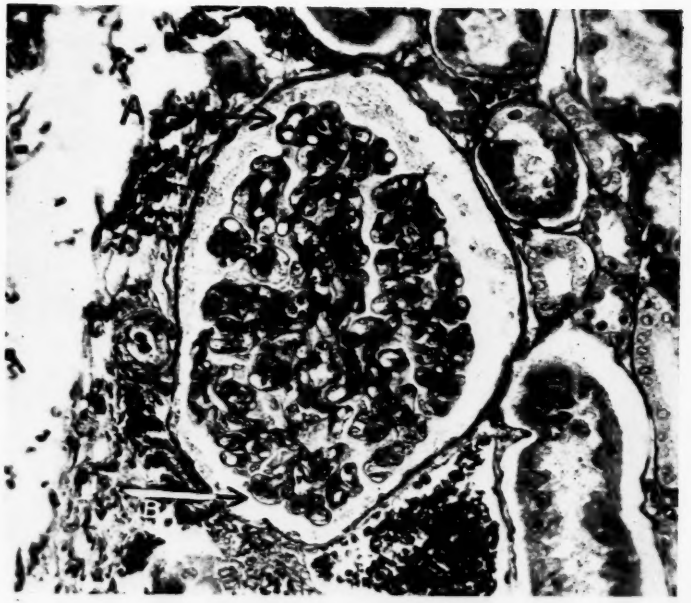


FIG. 5.

Kidney. (Microphotograph × 240, Formol-Zenker, McGregor's Azan-carmine.) A typical glomerulus most of whose capillaries show thickening of their basement-membrane. A good example is marked by arrow A, contrasting with a normal capillary marked by arrow B.

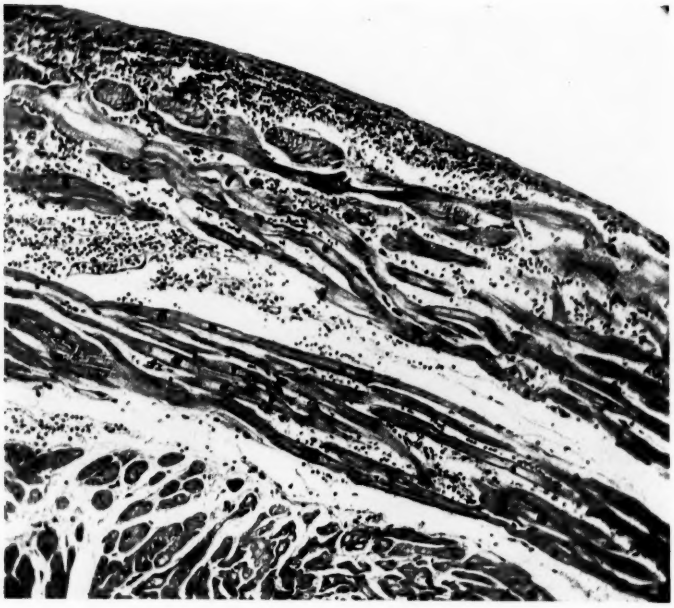

FIG. 6.

Heart. (Microphotograph × 122, Formol-Zenker, Haematoxylin and Eosin.) Swollen degenerate subendocardial muscle-fibres separated by interstitial collections of red cells.

nephritis, leucaemia, haemophilia and hypertension by Halban and Seitz.[3] Läwen,[4] in a general article on massive retroperitoneal haemorrhage, states that it may occur in eclampsia, although he does not actually describe any cases. Our pathological findings of eclampsia on the one hand and the absence of any rupture of large vessels on the other point, therefore, to the retroperitoneal capillaries as to the source of haemorrhage. This tendency to capillary thrombosis and haemorrhage in eclampsia is, of course, well exemplified by the frequent occurrence of such lesions in the brain, liver and placental site.

It is difficult to neglect the clearcut clinical picture of trauma—the sharp onset of pain in the abdomen during coitus, the collapse, the swift demise. Trauma may well have played the part of precipitating the haemorrhage. Low[5] has recently described a similar case of fatal retroperitoneal haemorrhage in late pregnancy which he ascribed to the strain of defaecation. Constipation was a common factor in most of the cases of spontaneous retroperitoneal haemorrhage in men and non-pregnant women reviewed by Eiss.[6] A personal communication by an ex-colleague, Doctor Eileen Wilson, M.R.C.O.G.,[7] tells of

"A woman aged 25, without toxaemia of pregnancy, admitted in labour to Epsom County Hospital in 1942. A full-time child was born normally after 8 hours. Twenty-six hours later, the woman collapsed suddenly with signs of internal haemorrhage and a mass was felt in the left flank from pelvis to costal margin extending across to the right flank. Blood transfusion did not effect improvement. Laparotomy was done 20 hours after the onset of symptoms and a great clot removed from the retroperitoneal space, but further bleeding began and the woman died at once. The haemorrhage was found to have come from a ruptured aneurysm of the left renal artery."


## Summary.

A case is described of massive retroperitoneal haemorrhage causing death in a woman with pre-eclamptic toxaemia in late pregnancy and following coitus. Eclamptic lesions were found in liver, kidneys and heart. The source of the haemorrhage was not discovered. The literature is reviewed.



## Acknowledgments.

We are indebted to Mr. E. V. Willmott, A.R.P.S., for the photographs and to Mr. J. R. Baker, A.I.M.L.T., and Mr. J. C. Griffin, A.I.M.L.T., for the histological preparations. We would like to thank Dr. Eileen Wilson for the description of Dr. Brown's case and for permission to publish it here.


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# A Series of Forty Cases of Vesicovaginal Fistula

BY

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THE problem of the vesicovaginal fistula has interested gynaecologists ever since surgery began to offer hopes of its cure. Mafouz Bey[1] has given a fine review of the history of its treatment, and Chassar Moir[2] has retold in detail the fascinating story of J. Marion Sims's work on this condition. At one time a terrible problem by reason of its frequency and the distress which it causes, the vesicovaginal fistula excites interest in western countries nowadays largely by its rarity and its historical associations. The difficulties of treating those cases which are encountered remain.

Where good obstetric practice is not widely available these fistulae are still fairly common. In the last 6 years I have had 40 cases admitted under my care. The series is not large compared with those that many surgeons in the East could produce —Mafouz Bey himself had treated more than 400 by 1938—but it may serve to illustrate some of the types which occur, and the results obtained, for the most part, by the flap-splitting operation. The aetiology of the obstetric fistula and the preliminary treatment have been described by many authors, and no further mention of either is therefore necessary in this paper, except to stress that this preliminary treatment is most important (anaemia and urinary sepsis are the conditions most requiring attention) and to mention that in this series one fistula was the result of a radium burn, and another the result of lympho-granuloma, which caused the whole of the urethra to slough.

I find it convenient to classify vesicovaginal fistulae according to their situation, and under three heads. First, a fistula may lie in the vagina at an appreciable distance from the cervix and from the internal urinary meatus: a midvaginal fistula. Or it may be adjacent to the cervix, when scar tissue usually fixes it to that structure: a juxta-cervical fistula. Or it may be near to, or actually involve, the internal urinary meatus or even the urethra, a part or whole of which may have sloughed away. This it is convenient to call a juxta-urethral fistula.

In this series of 40 cases there were 18 juxta-cervical, 5 midvaginal, and 17 juxta-urethral fistulae.

All the juxta-cervical and midvaginal fistulae were operable, but 2 of the juxta-cervical cases were unfit for operation on account of their poor general condition. They went home and unfortunately did not return. The remaining 16 juxta-cervical fistulae and all of the 5 midvaginal were operated upon by the flap-splitting method. Two of each type had been operated upon before elsewhere, once or more than once. All were healed at the first attempt except 1 juxta-cervical fistula. It was not a difficult case but the patient pulled out her catheter and a pin-hole fistula remained which may have healed since, but I have been unable to trace her. The midvaginal

fistulae were most of them comparatively easy, but many of the juxta-cervical were most difficult to reach or to suture, on account of the immobility of the cervix and of bands of scar tissue which narrowed the vagina. Nevertheless, there was no case which I thought would be better approached by opening the abdomen or by opening the bladder suprapubically, and I begin to doubt whether it is often necessary to do so.

The juxta-urethral fistula were much more difficult. There were 17 cases of this type, of which 3, being unfit until the local or general condition improved, left hospital and unfortunately did not return. Of the 14 cases which remained, 2 were regarded as inoperable because in each case a large part of the inferior border of the fistula was bone: the os pubis; and it seemed, to me at least, impossible to mobilize the bladder. In the whole series there has been no other criterion of inoperability. Success was not achieved in 1 of these cases in which there was also an inoperable recto-vaginal fistula, and the other case died after transplantation of ureters from suppression of urine. Seven cases were treated by the flap method, and 5 were cured by the first attempt. One of the failures was still operable but did not return, and the other died from suppression of urine after transplantation of ureters. For each of 3 cases in which the whole of the urethra had sloughed a new urethra was made. One of these was very successful, but the other 2 became very septic and sloughed. One of them was not an obstetric fistula but the result of lympho-granuloma, and it was perhaps a mistake to attempt it, but the tissues seemed healthy and the operation carried little or no risk. The other of these failures had one ureter transplanted successfully but the patient refused to wait for the second operation, and she too must, therefore, be classed as a failure. Two cases remain to be accounted for, and in each the "saccule" operation was performed, as described below. It failed each time, but in one of the cases both ureters were successfully transplanted, and the other case was made operable by the flap method and was thereby cured.

To sum up, 21 juxta-cervical and mid-vaginal cases submitted to operation, and 20 were cured by the flap method at the first attempt. In 14 juxta-urethral cases the patient agreed to operation. The flap method was performed for 7, with 5 successes. Three more patients were cured by other methods, making a total of 8 cures, if we may call transplantation of ureters a cure, which, strictly speaking, it is not, since the fistula remains. Combining all types: of 40 cases, 35 submitted to treatment and 28 were cured, 25 by the flap method and 3 by other means. There were 2 deaths from suppression of urine following transplantation of ureters. Two cases were regarded as inoperable, and these cases are included in the 35 which submitted to treatment. Only 4 patients submitted to a second operation: one was an unsuccessful saccule operation cured subsequently by the flap method, and the other 3 underwent transplantation of ureters.

I propose to describe the flap operation in some detail since this was used most frequently and with most success.

### The Flap Operation.

Crossen[3] classifies the various methods of operation which have been used. He recognizes two methods of flap-splitting, one from the margin of the fistula outwards towards healthy tissue, and one from healthy tissue towards the fistula. This latter method seems to be little mentioned in the literature, and little used as far as my enquiries have informed me, yet it seems to me to be immeasurably the sounder. To try to split the margins of the fistula into

vaginal and vesical flaps is to make unnecessary difficulties. It is very hard to find the plane of cleavage (the pubo-cervical fascia) by searching for it through the scar tissue of which the margin of the fistula is composed. It is, on the other hand, very easy to find it at some place remote from the fistula where there is no scarring. One makes an incision through the vaginal skin and then inserts into it a closed pair of blunt-pointed scissors, which easily finds the correct fascial layer, just as in colporrhaphy. The further procedure is described below, but it remains to consider other difficulties which dissection from the edge of the fistula brings about. First, there is the difficulty of inserting the knife into the margin of the fistula. It may be inserted into the superior margin more or less accurately and parallel with the wall of the vagina, and one may hope that it will find the pubo-cervical fascia. But it is impossible to insert the knife into the inferior margin of the fistula parallel to the vaginal wall, unless there is a marked genital prolapse and the vaginal wall can be pulled outside the ostium. One will rarely be so lucky: as a general rule the fistula is difficult to reach and genital prolapse is extremely rare, no doubt owing to the dense scarring of the vagina which is very common. But having attempted the incision there is still the difficulty of separating the vagina from the bladder. The edge of the fistula is very liable to be bruised and lacerated in the attempt, yet it is this very edge which one is to suture presently and upon which one will rely for healing. The approach described below preserves this edge from trauma and enables one to mobilize the bladder with comparative ease.

*Time of operation.* I prefer not less than 3 months to elapse from the last labour or last operation before a repair is attempted. If the tissues are at all oedematous they will tear as one dissects and the sutures will not hold.

*The anaesthetic.* An intraspinal anaesthetic of light percaine is ideal and lasts long—a point of importance as a difficult case may take from 2 to 3 hours to complete.

*The position and exposure.* I have only experience of the lithotomy position. It may be necessary to pull the patient very far down, particularly when operating upon a juxta-urethral fistula. Such fistulae are often pulled upwards and around the symphysis away from the operator, and this peculiar retraction around the symphysis makes some of them very hard to reach. In such cases the patient should be pulled as far down as possible (supple Indian joints are a great help here) and then the head of the table should be lowered. It is thus possible to make the anterior vaginal wall almost perpendicular to the surgeon's line of vision and the fistula can better be seen. This manoeuvre causes the perineum to rise and obscure the view, but a paravaginal incision will remedy this. One will often have to resort to this incision for all types of fistula; one should employ it to gain free exposure without hesitation. Bands of scar tissue in the vagina may also obscure the view, particularly in high fistulae, but one needs to think carefully where these can be divided with the greatest advantage. It is desirable to cover the repair in the bladder with vaginal skin, and it is a mistake to incise a band of scar tissue in the wrong place and to find that there is very little viable vaginal skin in the vicinity of the repair from which to fashion a flap. Generally speaking it is safe to divide scar tissue on the posterior vaginal wall, and it is usually convenient if the paravaginal incision is carried up to these bands and through them.

An Auvard's speculum is usually re-

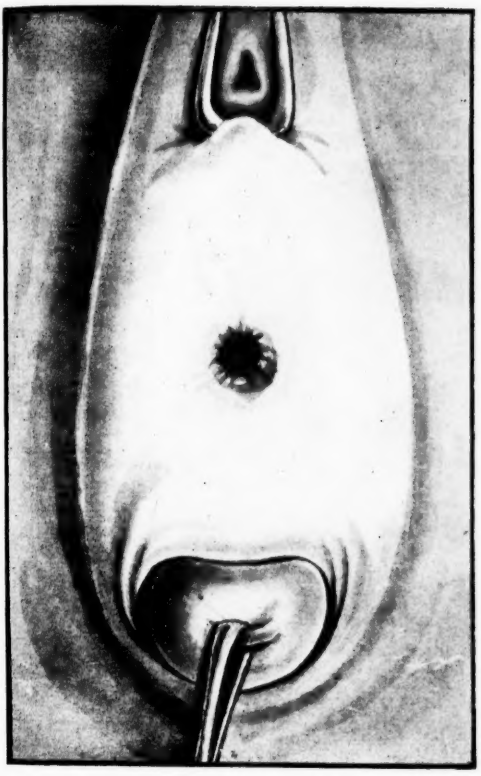

FIG. 1.

An easy midvaginal fistula.

FIG. 2.

Both incisions have been made, and the scissors are separating the bladder from the vagina. In practice this is done first from the other (i.e. higher) incision, so that any blood which issues from it will not obscure one's work on the lower incision.

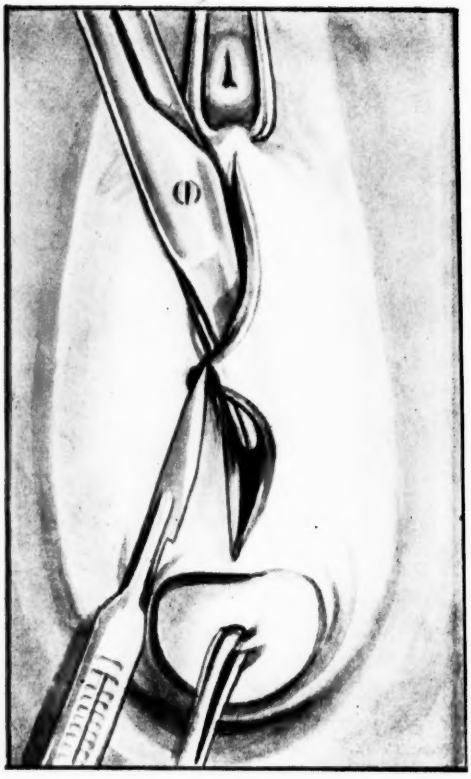

FIG. 3.

Bladder and vagina have been separated widely through each incision on each side. Only the edge of the fistula now holds them together. A No. 11 Bard-Parker knife is dividing this last connexion, cutting on to a pair of closed scissors inserted through the lower incision and emerging from the upper.

G.B.T.

FIG. 4.

The fistula is free and the bladder has been mobilized as far as is desired. The vaginal flaps are held back by tissue forceps. Traction sutures hold the bladder steady, and the suturing of the fistula has begun. In practice the suture is held at the correct tension by an assistant, but this has not been shown.

FIG. 5.

The suturing is complete and the traction sutures have been removed. It remains only to suture the anterior vaginal wall.

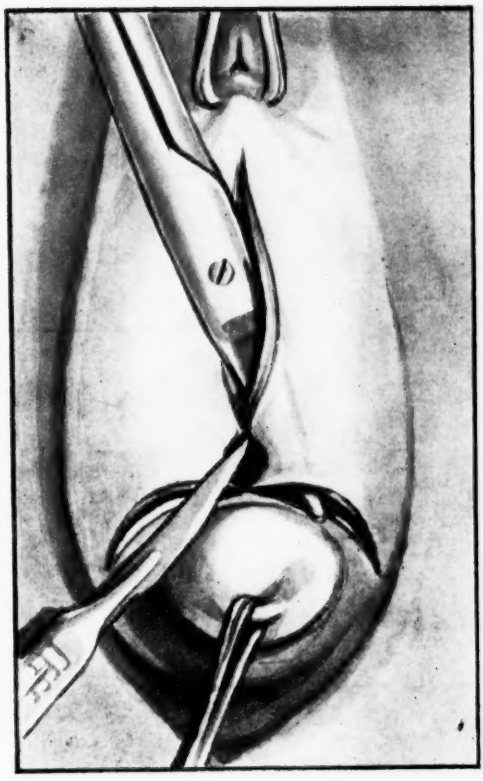

FIG. 6.

The procedure adopted for juxta-cervical fistulae. After retracting both vaginal flaps, the bladder, with the fistula, will be dissected from the front of the cervix. This dissection may also be required in the lower fistulae (midvaginal or juxta-urethral).

quired, and sometimes it is helpful to sew the labia minora to the buttocks to keep them out of the way and to open up the ostium. Finally, a volsellum is usually applied to the cervix and a tissue forceps near the lower end of the vaginal wall so that the anterior vaginal wall may be kept on the stretch while the first incision is being made. More often than not, the volsellum will have to be pushed upwards (anatomically speaking, that is) in order to stretch the vagina, since the cervix cannot be pulled down if there is no genital prolapse.

*The incision.* An incision is made from below upwards to the fistula through the vaginal skin, and then from the fistula upwards to the cervix (Fig. 1), except in juxta-cervical fistulae, which will be dealt with separately. The depth of the incision is the same as for colporrhaphy, and there is no particular difficulty except that usually one is working within the vagina. The points of a closed pair of Mayo's scissors, curved on the flat, are then thrust into the fascial space between the bladder and the vagina, which are then easily separated by opening the blades of the scissors (Fig. 2), again as in colporrhaphy. This manoeuvre is carried out on each side through each of the incisions, so that finally the bladder and the vagina are held together only by the adherent margins of the fistula. All around, the bladder and the vagina have been cleanly separated in a plane of cleavage easily found by this approach from a distance, where scarring is minimal or absent. With juxta-cervical fistulae the incision above the fistula towards the cervix obviously cannot be made as just described. It is made transversely across the front of the cervix, between it and the fistula, and it is then curved on each side laterally towards the fornices (Fig. 6). This may be difficult on account of scar tissue, and in one case it simplified matters to carry the paravaginal incision right up to the fistula itself, after which separation of the lateral flap was much easier.

*Freeing the fistula.* A closed pair of scissors or a probe is now inserted in the inferior incision, and, if possible, made to emerge from the upper incision (Fig. 3). It is thus pressed against the scar tissue which still binds the bladder and vagina together at the margin of the fistula. A sharp knife (a No. 11 Bard Parker is very useful) is then used to cut cleanly through this margin on to the scissors or probe. This is done on each side, and thus the fistula is freed. The orifice in the bladder is clean-cut instead of lacerated, and it is fit for suturing. It has not been touched at all in the separation of the flaps.

In juxta-cervical fistulae and in the larger midvaginal ones, and even in some of the juxta-urethral, it is necessary to mobilize the bladder from the front of the cervix. This is a very common step in gynaecological operations and need not be described here. If more mobilization laterally is required, gauze dissection will supply it. The pull of the gauze is on the bladder far away from the fistula, which will not suffer any harm. It must be emphasized that adequate mobilization of the bladder, so that the fistula may be closed without any tension whatever, is absolutely essential.

*Suturing the bladder.* As a general rule it is easier to suture the bladder horizontally, since by this means the best advantage may be taken of the loose bladder which has been dissected from the cervix. It is useful to insert 2 traction sutures, one at each side of the orifice, not less than a $\frac{1}{4}$ inch from its edge. A gentle pull on these will prevent the loose bladder from falling away, and will keep the fistula steady and as near to the operator as possible (Fig. 4).

I have used only very fine chromicized catgut, on atraumatic needles where there was room to use them, and closed the gap with a simple continuous suture, about 12

stitches to the inch. After each stitch the catgut should be gently pulled to the right tension and kept at this tension by the assistant. If it is allowed to slacken then there is great danger of the urine leaking at this point. This may be detected before the end of the operation, but if several other sutures have been inserted each of them will have to be tightened, and in doing so one or more of them may tear out. In any case there will be the risk of fraying the edge of the fistula, which it is one's particular care to preserve from trauma. I believe it to be very important also to start and to finish the suture at least $\frac{1}{8}$ inch from the edge of the fistula, so that there may be no leakage from the ends of the suture line. The suture should pass through the bladder musculature down to, but not through, the mucosa. This, it must be admitted, is in many cases but a counsel of perfection. When the fistula is hard to reach it may be most difficult to pass a suture at all, in which case a good bite of bladder muscle probably suffices. This is certainly better than going too far and including the bladder mucosa, which invites a leak. If the mucosa prolapses through the fistula so that it is hard to avoid piercing it with the needle, then it may be trimmed with scissors. Sometimes after the suture line is completed there remains plenty of loose bladder wall, and there is little difficulty in passing a second suture line Lembert fashion. If, however, the bladder wall is thin, or there is not enough left to allow of easy infolding, then it is better to leave well alone. The infolding suture is certainly not essential and I have often omitted it.

Sometimes the margins of the fistula are so far out laterally that the traction sutures referred to above cannot be used, and often it is difficult even to pass a suture to close the gap. Very small needles have to be used. Frequently atraumatic sutures are far too big, and there is too little space to manoeuvre them in. Occasionally it helps to employ 2 stitches, one passed from each side while the central portion of the hole is still open. These 2 sutures meet in the middle and are there tied together. By this means the act of suturing becomes easier as one progresses towards the middle.

It is hard to exaggerate how difficult this part of the operation may be. Several trials may be necessary, before each stitch is passed, to adjust the needle in the needle-holder so that it may be introduced up as far as the fistula and correctly inserted. The concentration necessary to do this with the least possible trauma and with great accuracy makes the operation often most fatiguing. And indeed throughout the operation each movement must be deliberate; even a little obscuring blood clot must be removed with care, for a slight tear in the bladder wall or the pulling out of a stitch may ruin the operation.

*Testing the suture line.* The bladder is now filled through a rubber catheter with methylene blue under a pressure of 1 or 2 feet of water. The catheter may usually be a No. 10 or 12, but a smaller one is often necessary for juxta-urethral fistulae, as a larger one may strain the suture line and cause a leak. If there is no leak, the bladder is emptied and the catheter turned to one side. Only once did I find the suture line leaking. It had been particularly difficult to sew the left extremity of the fistula which extended almost to the pubic ramus. I passed a purse-string suture around the leaking area and the repair healed, but I am bound to say that I think I was fortunate.

*Suturing the vagina.* Often this is fairly easy and sometimes one can usefully excise a portion of the vaginal flaps, as in colporrhaphy. In case of doubt it is better not to do so, because if the operation is a failure one will need all tissue possible for

the next attempt. There may sometimes be difficulty in making the vaginal flaps meet to cover over the repair in the bladder. Only on one occasion did I have to fashion flaps and bring them across from their original position, and the operation was a failure. On another occasion I had to leave the bladder uncovered after performing a saccule operation, and this operation also failed, although the fistula was cured subsequently. Having sutured the vagina and also the paravaginal incision, if one has been necessary, one sutures the catheter to the labia minora. The patient does not experience any pain from this unless she tries to pull the catheter out, and she soon desists. Self-retaining catheters I have not used. They easily fall out, they are painful if their shoulders press on the trigone, and for juxta-urethral fistulae they may press on the suture line and strain it.

The vagina is now gently but firmly packed with gauze soaked in 1 in 1,000 acriflavine, which is removed after 24 hours.

*After care.* The main point of the after-care is to ensure that the bladder remains empty. It is therefore necessary to see that the patient does not pull out the catheter and that the catheter does not become blocked with blood clot, mucus, or phosphatic deposits. Quite a little blood may flow into the bladder through the fistula in the course of the dissection, and it is wise to clean it out before the bladder is closed. Even so, a little may be left and give trouble. For 12 days the drainage by catheter continues, and every hour, day and night, the nurse records the amount of urine which has drained. One thereby receives early warning if the catheter becomes blocked. Should it do so the bladder may fill up and strain the suture line sufficiently to make it leak. But if the block has existed for no more than one hour, then little urine will have collected in the bladder in this short time. The catheter may be cleared by injecting up it 5 to 10 c.c. of sterile water; occasionally it may be necessary to change it. This must be done with particular care if the fistula was juxta-urethral.

My patients are nursed on their backs, but if they find this very irksome they are permitted to turn on their sides with care after 4 or 5 days. I doubt very much if nursing patients in the prone position, as some authorities recommend, can prevent the urine wetting the suture line. The anterior and posterior walls of the bladder are in contact as long as the urine is being constantly drained by catheter, and the posterior wall must, therefore, be just as wet when the patient is prone as when she is supine. Even if a suprapubic cystostomy were made the bladder walls would collapse and touch each other, and the urine is bound to wet them both. I do not think it is at all necessary to keep the suture line dry, nor do I think it possible to do so. Gastro-enterostomies heal well yet are never dry, and nobody ever attempts to keep them so, though I must admit that the comparison is not quite fair as gastro-enterostomies would probably not heal as well but for the sealing action of the peritoneum. Howbeit, it is hard to reconcile the success of the juxta-cervical fistulae in this series with the need for keeping the suture line dry. The failures in this series were, all but one, juxta-urethral fistulae, which were no wetter than the others. The failures I ascribe to difficulties in suturing and to the thinness of the urethra from which in some cases, I feel certain, the sutures cut out, despite great care to avoid tension.

Some surgeons, at the other extreme, order daily bladder wash-outs, but this practice I abandoned after my first few cases except when the catheter became continually blocked with deposits. It seems

to be unnecessary as a routine; copious drinks are effective in keeping the urine dilute, and urinary antiseptics may also be given.

The less interference the patients undergo the better, and for this reason the bowels are left alone for 10 days, before which time it is rare for them to act spontaneously. Patients do not suffer any harm or discomfort from this, and they pass a stool without difficulty when given an enema. On the 12th day the catheter is removed, and the patient is then encouraged to pass water every 2 hours for 5 or 6 days, and thereafter only when she has the inclination. It is striking how rapidly a patient begins to control her bladder, and how soon the bladder stretches to accommodate a normal amount of urine, although it has been empty and contracted for the months or years during which it has leaked from the fistula.

One final point regarding the after-treatment: one need not be unduly despondent if the patient appears to be leaking. Very often the leak is around the catheter and the repair has not broken down. One must, however, make certain that the catheter is not blocked.

After the operation patients are kept in bed for 4 weeks, and advised to avoid coitus for a further 3 months and to report early if they again become pregnant.

## Other Operations Used.

1. *Bonney's Operation for Elongation of the Urethra.*

This operation of Bonney[4] was adapted for 3 cases. In these there was no urethra left. It had sloughed away leaving a hole leading from the bladder directly into the vagina at the site of the internal urinary meatus. Only in 1 case was the operation successful; in the other 2 sepsis spoiled what appeared to be a sound operation. Despite these 2 failures I feel sure that this is the ideal operation for such cases.

2. *Saccule Operation.*

This was used in 2 cases. In these the upper part of the urethra had sloughed away and there was a considerable gap between the fistula and the remains of the urethra, too big a gap to bridge by anastomosing the fistula to the top of the urethra. The aim of this operation is to turn over flaps of vaginal skin and to suture them so that they form a saccule intervening between the fistula and the urethra. The urine should flow from the fistula into the saccule and thence out through the urethra. On both occasions the saccule failed to heal, but in 1 case only a small portion remained open. This small hole (about $\frac{1}{4}$ inch in diameter) it was possible to close by the flap operation, and the patient was thereby cured.

3. *Colpocleisis.*

This unpleasant operation was attempted twice in the same patient and it failed each time. I doubt if the operation is justified unless, as in this case, there were an inoperable vesico-vaginal fistula and an inoperable recto-vaginal fistula. I thought a colpocleisis would be better than leaving the patient untreated, and she herself was of the same opinion. The tissues were like cartilage, and several attempts to cure the recto-vaginal fistula and 2 attempts thereafter to perform colpocleisis were complete failures.

4. *Transplantation of Ureters.*

This paper is not concerned with the technique of this operation, which has been well described by others. My comparatively small experience of vesicovaginal fistulae, however, leads me to think that only a small percentage of them needs transplantation, and that when a large series of transplanta-

tions is described one should enquire what percentage of the total number of fistulae seen the series represents. Too frequent performance of this operation is to be deplored. The mortality-rate is bound to be higher than in the repair operation: Murray and Ahmed[5] report a mortality-rate of 21.5 per cent. The result is nothing like so satisfactory as a repair, and further it is not a cure of the fistula, although it certainly gives relief.

### Healed Fistula and Subsequent Deliveries.

Not all fistulae, by any means, are due to disproportion. Nevertheless, I feel that all subsequent deliveries, if circumstances permit, should be by classical Caesarean section. In a vaginal delivery the descent of the head is bound to stretch the scar of a healed fistula, and the risk of its tearing must be considerable. Caesarean section does not seem too big a price to pay as an insurance against so unfortunate an occurrence. Even Case No. 23, in which a natural delivery took place within 2 hours of labour starting without any difficulty at all, does not alter this opinion. If difficulties should arise in a vaginal delivery the fistula may very likely recur; when they have arisen, classical Caesarean section may avoid these difficulties, but it may well be too late to perform this operation safely. The lower segment operation is the operation of choice if labour has been in progress for more than a few hours, and at any time if the membranes have ruptured. But in peeling the bladder from the front of the cervix one may easily tear the scar of the healed fistula, especially if it is adherent to the cervix. But might one attempt vaginal delivery if the fistula had been juxta-urethral, since the lower segment incision will not encroach so far down? Again I think not, because if nevertheless the juxta-urethral fistula should recur the chances that it can again be repaired are much less than the chances of success for the higher fistula: some 57 per cent in this series as against some 95 per cent. Few of those who have suffered the horrors of a fistula would not share this view and be unwilling to have a Caesarean section; still fewer surgeons who have repaired a fistula would wish to see it subjected to the risks of a vaginal delivery.

It seems justifiable, therefore, to deliver by classical Caesarean section in all cases which one sees before labour begins and when considerations of sepsis do not prohibit. In the majority of cases there will probably be no question of allowing a natural delivery as the cervix and vagina will be too densely scarred.

### Summary.

1. Vesicovaginal fistula are classified, according to their situation, as juxta-cervical, midvaginal, or juxta-urethral. In the 40 cases there were 18 juxta-cervical, 5 midvaginal, and 17 juxta-urethral fistulae.

2. The results of their treatment are given, and it is shown that the juxta-cervical and midvaginal fistulae were all operable by the flap-splitting method, and that all but one which were operated upon healed at the first attempt.

By contrast the juxta-urethral fistulae were less successful, only 8 being cured out of 14 cases which submitted to operation.

3. A technique for the flap-splitting operation is described and illustrated, and other operations used and the after-treatment are also described.

4. The view is advanced that the repair by the abdominal route is probably only rarely necessary. It was not required once in this series.

5. It is suggested that transplantation of

the ureters should rarely be required and should be avoided if possible.

6. Classical Caesarean section in suitable circumstances is advocated in cases of pregnancy following a successful repair.

I wish to thank the medical officers who have assisted me, particularly Dr. E. V. Kalyani, M.D., who has helped at most of the operations and taken charge of the after-care in all cases; and Mr. P. M. Ratnasabapathi who has drawn the illustrations.

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# Vesicovaginal Fistula

## A Review of 42 Cases treated by Plastic Repair or Ureterosigmoidostomy

BY

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THOSE who have seen cases of vesicovaginal fistula all testify to the state of utter misery of the patient. Constantly wet and stinking, she is ashamed to go out and is a nuisance in the house—not only is she objectionable to others but has herself to endure the odour, wetness and resulting soreness. In India she is often deserted by her husband and unwanted in her parents' home. She is unable to support herself except perhaps by begging.

### CAUSE.

In countries where vesicovaginal fistula is a common condition the most usual cause is faulty midwifery. Including the cases in this series I have seen altogether about 80. Of these the cause was malignant disease in 3 cases (cervix 2, bladder 1) and 1 occurred after total hysterectomy. In the remainder, that is, about 95 per cent, the cause was obstetrical. Murray and Ahmed[1] in Calcutta found that in 65 inoperable cases the cause in 51 was obstetrical.

But in countries with a good midwifery service the picture changes, and the condition not only becomes a rarity but the cause differs. Operation, especially total hysterectomy, and radium applied to carcinoma of the cervix, are the chief factors. Latzko,[2] reviewing 97 cases in America over 30 years, finds 65 occurring postoperatively, 28 due to obstetrics and 4 to other causes. Miller[3] found 70 per cent in another series to be due to operation.

But, taken as a whole, these postoperative cases are relatively infrequent and the outstanding cause of vesicovaginal fistula is bad obstetrics. Many factors contribute to this cause in a country such as India. In many areas there is a lack of adequate facilities, doctors, nurses, hospitals, antenatal care. Bad communications make it difficult for a patient to be taken to hospital or for a doctor to reach her home. The Mahommedan religion will not permit a woman to be treated by a man doctor, and this edict is still strictly observed by many millions of Muslims and some Hindu castes. Custom dictates that midwifery be handled by the native "midwives" or *dais*, ignorant women who have learnt their craft from tradition. They are called in when labour begins, do not allow the patient to rest or take nourishment, and insert a dirty hand into the vagina with nearly every pain. Many women, especially primigravidae, become utterly exhausted and are unable to deliver themselves. Their condition deteriorates, the *dais* become alarmed and medical aid is sought so that the responsibility may be shifted. In a typical case one sees an exhausted patient, with the uterus approaching tonic contraction, the head has been showing at the vulva for many hours, and there is a greatly distended bladder. The soft parts have often been abraded.

### PATHOLOGY

There is no doubt that in the actual causation of a fistula prolonged pressure of the

head on the soft parts with cutting off of the blood supply and subsequent necrosis is the most important factor, especially if the bladder is distended. In most cases the forceps have to be used to complete delivery, and in some instances careless use and trauma may be contributing causes of the fistula. In such cases the leak of urine may occur at once; but the fact that it usually does not appear till the 3rd or 4th day points to a slow destruction of the vesicovaginal septum by necrosis, and this is more often due to prolonged pressure than sudden trauma. In delivering such cases it is important to perforate the head (the foetus is dead in nearly every case) in order to diminish its size if there is any difficulty, otherwise force in traction may be needed and this should be avoided.

In Africa the custom of female circumcision with resulting scarring which delays delivery is thought by Roberts[4] to be a cause of fistula, but this is doubted by Preston[5] who considers delay with pressure to be the most important factor. This custom is unknown among Indian women, and minor degrees of disproportion together with the exhaustion induced by the malpractices of the *dais* lead to the delay which actually causes the fistula.

Prevention will be by improving and increasing the midwifery service, by education and breakdown of superstition and ignorance and by improved roads and transport facilities. In many of the cities of India these conditions already obtain, and thousands of women are safely delivered yearly in the hospitals and nursing homes. The problem for rural India still needs to be tackled.

## Cases in this Series.

In the five years from March 1938 to March 1943, I treated personally the 42 cases of vesicovaginal fistula now under review. In all these the fistula had occurred after labour, 33 after the 1st delivery and 9 after 2nd or later deliveries. Some patients were in good condition, others ill-nourished from poverty and various infections. Preliminary treatment was given when necessary, especially for intestinal worms, ascaris and ancylostome, and urinary infection. Most of the patients were young women, half being under 25 (Table I), and in more than two-thirds of the cases when the duration of the fistula could be ascertained, the age at which it had occurred was under 25 (Table II).

Table I.
*Age of Patient when seen.*

| Age | No. of patients. |
|---|---|
| 17 to 19 ... ... ... ... | 7 |
| 20 to 24 ... ... ... ... | 15 |
| 25 to 29 ... ... ... ... | 6 |
| 30 ... ... ... ... ... | 14 |

Table II.
*Age of Patient when Fistula Occurred.*

| Age | No. of patients. |
|---|---|
| 15 to 19 ... ... ... ... | 10 |
| 20 to 24 ... ... ... ... | 10 |
| 25 to 29 ... ... ... ... | 4 |
| 30 ... ... ... ... ... | 10 |
| Not stated ... ... ... | 8 |

## Obstetrical History.

In 24 patients a history of the labour was obtained. Three stated that labour had been unduly prolonged, but ended spontaneously, in 12 the labour was terminated instrumentally after much delay, and in 9 there was a history of delivery by the forceps without mention of delay, though it had almost certainly occurred. In 5 cases incontinence of urine had occurred within 24 hours, in 12 cases between the 3rd and 5th days, and in 7

cases later. Two patients had partial control on lying down, but the rest were completely incontinent.

Infection of the bladder was found in 18 cases, in 2 appearing for the first time after operation, the urine not giving any growth on culture beforehand. In all cases Bact. coli was present, in 5 gonococcal infection also, and in 2 others the infection was mixed. Eight cases were free from infection throughout. In the remainder (17) a note was not made, but as urinary examination was done as a routine these cases were probably not infected. In all infected or doubtful cases preliminary treatment, usually with sulphonamides, was given.

### Condition of Vagina and Nature of Fistula.

One patient had a rather lax vagina with a degree of cystocoele. In all other cases the vagina was normal in size or narrowed with some degree of scarring. Special note was made of scarring in 11 cases. The size of the hole was pinpoint in 2 cases; small (not more than $\frac{1}{2}$ inch in any diameter) in 8 cases; of medium size ($\frac{1}{2}$ inch plus) in 8 cases; of medium size and at the neck of the bladder in 7 cases; large or very large in 15 cases; and slit-like and situated high up behind the symphysis in 2 cases. In these 2 last cases, and in 7 of those classed as large, the urethra was partially or completely destroyed.

### Treatment.

A. *Plastic Repair.*

Most vesicovaginal fistulae can be repaired by plastic methods approaching from the vagina.

The important points in performing the operation are:—

(1) Preliminary clearing up of infection.
(2) Excision of scar tissue.
(3) Mobilization of the bladder wall.
(4) Closure of the fistula without tension.
(5) Haemostasis.
(6) Drainage of the bladder to prevent over-distension and strain on the repair site.

The fistula had not been present in any case less than 4 months (the ideal time to operate is usually considered to be about 2 months after occurrence), and in some it was of 10 years duration (Table III). Seven patients had been operated on previously in other hospitals, 2 having had 2 operations each.

Table III.

*Duration of Fistula when first seen.*

| Duration | No. of patients |
|---|---|
| 4 to 6 months ... ... | 7 |
| 6 to 12 months ... ... | 8 |
| 1 year ... ... ... ... | 9 |
| 2 to 5 years ... ... ... | 5 |
| 5 to 10 years ... ... ... | 2 |
| Over 10 years ... ... ... | 3 |
| Not stated ... ... ... | 8 |

### Complications.

Of the patients 3 had a rectovaginal fistula in addition and 1 a complete tear of the perineum with faecal incontinence.

*Technique.*

With the patient in the lithotomy position and anaesthetized, the largest possible speculum is placed in the vagina. The labia are retracted and the fistula exposed. An incision is made in the vaginal mucosa encircling the fistula and leaving a narrow rim of scar tissue attached to the bladder wall. The flaps of vaginal mucosa are then dissected upwards and downwards respectively freeing the bladder wall for $\frac{3}{4}$ inch all round. The greatest difficulty may be experienced at the angles of the fistula which may be deep and inaccessible. In such cases it is helpful to extend the incision on to the lateral vaginal walls for it is essential to reach the limits of the fistula.

The edge of the fistula is pared and haemostasis secured, using fine silk ligatures. Interrupted stitches of No. 00 catgut on a small round-bodied needle are then inserted into the bladder wall. These pick up the muscle layer avoiding the mucosa and invert the edges of the fistula. Great care is needed at the angles of the fistula to make the closure water-tight. Finally the vaginal layer is closed with No. 1 catgut. A No. 12 catheter is tied in and continuous drainage of urine maintained. Daily small bladder washes are given. The patient is nursed either prone or supine. The catheter is removed between the 10th and 14th days and the patient encouraged to pass urine frequently. After 2 or 3 days she is allowed to sit up in bed and gets out of bed a few days later.

In all, 27 cases were treated by attempts at plastic repair, being considered operable. The results are shown in Table IV.

These results can be compared with those obtained in the Calcutta Medical College,[6] when the figures in 104 cases were:—

Cured, 55.8 per cent; failed, 40.4 per cent; died, 3.8 per cent.

TABLE IV.

| No. of cases | | Cured No. | Cured Per cent | Improved† No. | Improved† Per cent | Failed No. | Failed Per cent | Died* No. | Died* Per cent |
|---|---|---|---|---|---|---|---|---|---|
| 1st Group, 1938–39 | 12 | 5 | 41.7 | 3 | 25 | 3 | 25 | 1 | 8.3 |
| 2nd Group, 1940–43 | 15 | 12 | 80 | 0 | — | 3 | 20 | 0 | — |
| Total | 27 | 17 | 63 | 3 | 11 | 6 | 22 | 1 | 4 |

## CAUSES OF INOPERABILITY AND FAILURE.

*Size* is not necessarily a contra-indication to operation. Murray and Ahmed[1] state that a pinpoint fistula or one larger than a shilling is inoperable, but most surgeons would disagree with this (Chassar Moir[7]; Scudder[8]). In this series 1 pinpoint fistula out of 2 was cured, and 3 out of the 4 which were at least 1 inch in diameter healed.

*Difficulty of approach* makes some cases inoperable and this may be due to a very contracted vagina or an inaccessible fistula high up near the cervix in a lateral fornix or behind the symphysis pubis. Some of these cases can be approached by transperitoneal or transvesical routes but of these operations I have not had experience.

*Destruction of the urethra* is a cause of inoperability, as lack of proper sphincter control leaves the patient incontinent even though the fistula may be reparable.

*Gross destruction of tissue*, especially of the bladder wall, prevents repair in a few cases.

*Fistulae at the neck of the bladder* with the urethra torn away are difficult but may be treated successfully. Earlier attempts at 3 such cases failed, but in the last 3 seen a cure was effected.

## TECHNIQUE.

The problem in these cases is that a large hole in the bladder has to be joined to the much smaller urethra. The incision is made as before but an extension in the midline towards the meatus for ½ to ¾ inch is added. This allows better exposure of the urethra. Stitches are then inserted to narrow the bladder gap and then approximate the

* The 1 death occurred in a patient who was treated for a month before operation for malaria, ancylostomaises, anaemia and Bact. coli urinary infection, and who died on the 10th day after operation in a toxaemic state, having had a recurrence of the malaria.

† Partial control in certain positions.

narrowed gap to the urethra. I have found it best to pass these through all the layers of the urethra and then to pick up the bladder-wall, as the stitch often cuts out if one attempts to pick up only the muscle layer of the urethra, and there is loss of valuable tissue when the urethra is already shortened by partial destruction. The sutures closing the lateral extensions of the bladder gap may be interrupted or continuous. The rest of the operation and the postoperative care are as described above.

*Much scar tissue,* especially in the wall of the bladder, makes healing difficult. If the bladder wall can be mobilized and closed without tension success is likely (Miller,[3] Counseller[9]). It is not necessary to close the vaginal layer (Counseller[9]) and in 1 case in this series this was quite impossible. However the bladder was mobilized freely and closed in two layers, and the case was cured after a second small operation.

*Infection,* even though treated before operation, hinders success. Of 11 cases known to be infected and treated by plastic methods, 5 were cured, 5 failed and 1 died (of malaria and toxaemia), while in 16 cases not infected 12 were cured and 4 were failures.

*Postoperative haemorrhage* usually spells failure, though 1 of the 3 patients in this series, who had this complication eventually healed. That surgical experience and the acquiring of the necessary technique are important factors in obtaining success is evident from Table IV.

### Inoperable Vesicovaginal Fistula.

The most effective method of treating inoperable cases is by transplanting the ureters into the large bowel, usually the sigmoid colon. A number of techniques have been devised, some more suited to a 2-stage and some to a 1-stage operation. It is now generally accepted that oblique implantation is the best (Coffey No. 2 technique). Some surgeons consider it better not to leave anything in the lumen of the ureter (Grey Turner[10]), some use fine rubber tubing or a ureteric catheter and others a wick of catgut. More recently Higgins has devised a modification of the Coffey No. 3 technique and Hyams[11] has reported favourably on this method for cases of extrophy of bladder but I have not had experience of it.

In this series, 13 cases were treated by ureterosigmoidostomy, 9 in 2 stages and 4 at a single operation. In 6 patients an examination under anaesthesia performed some time earlier confirmed that the case was inoperable: in 7 patients this was not necessary.

In the 9 cases done in 2 stages the ureter was inserted obliquely, with a wick of catgut (No. 00) 1½ inches long left in the lumen. Of these, 6 were cured and 3 died, 1 after the first ureter only had been transplanted. Of the 4 cases treated at one operation, 2 recovered and 2 died. The technique was that used successfully by Sheppard.[12] Thus in the whole series there were 5 deaths in 13 unselected cases, a mortality of 38.5 per cent (cf. Murray and Ahmed,[1] with a mortality of 21.5 per cent in 65 selected cases).

In considering how the operation can be made safer much can be learnt from the cases of failure, and thus avoidable causes of catastrophe may be eliminated. Murray and Ahmed[1] admit that they selected only those patients who were in relatively good health. But it is very urgent to find a technique which can be used with reasonable safety on women whose general condition is not so good, but who implore one to relieve them of their misery. A 2-stage operation is usually safer (Green-Armytage[6]).

### Investigation of Cause of Death.

One of the 5 deaths was due to the anaesthetic (spinal percaine) at the 2nd operation. Another patient developed a urinary-

faecal fistula after the 1st operation, with amoebic dysentery (probably a recurrence of a chronic infection), and after the 2nd operation a retroperitoneal abscess due to leakage at the site of implantation which proved fatal. In this case the wall of the colon was very thin, as is often found in chronic dysentery, and operation was probably contra-indicated. A healthy colon wall seems to be essential.

The 3rd patient died after the 1st operation and postmortem showed that the final continuous suture enfolding the site of transplant had pierced the ureter wall, death being due to urinary peritonitis. This was carelessness in technique.

Of the deaths occurring after a 1-stage operation, 1 was due to renal failure, with anuria and uraemia. This was a case in which a 2-stage operation might have succeeded. The other was a young woman who made a good immediate recovery. She was well until the 16th day and died on the 22nd day after the operation. Death appeared to be due to peritonitis, but permission for postmortem examination even of the abdomen was refused. (I have also performed a postmortem on a patient operated on by another surgeon where death seemed to be due to kinking of the ureter at the site of insertion, with gross dilatation of the ureter and kidney pelvis, and destruction of kidney tissue.)

### Postoperative Complications.

Of the patients who recovered many had postoperative pyelitis and 1 had anuria with complete suppression for 2 days after the 2nd operation, but eventually recovered. Postoperative distension was troublesome in a number of cases. All the patients except 1 had good control of the rectal sphincter on discharge from hospital, and this last woman passed a few ounces of urine frequently, with sometimes a little leak between.

### Contra-indications to Transplantation of Ureter.

Ureterosigmoidostomy is useless if there is a rectovaginal fistula or a tear of the sphincter muscle of the anus. In these 42 patients 5 had one or other of these conditions. In 1, both fistulae were small and cured at operation. Another was cured of her rectovaginal fistula, but operation on the vesicovaginal fistula at the neck of the bladder failed. One patient had 2 large fistulae, of which the rectal one was successfully repaired after performing colostomy. The vesical fistula was inoperable, and the patient was discharged at her own request to come back later for transplantation of the ureters and closure of the colostomy but she was not seen again.

One patient had a complete tear of the perineum with an inoperable vesicovaginal fistula. When first seen the perineum and anal sphincter were repaired, and a year later both ureters were transplanted at one stage. She had complete control of urine in the rectum. One woman had a large vesicovaginal fistula and a tear of the perineum extending through the sphincter and up the rectum to the level of the cervix. There was great destruction of tissue, and neither bladder nor rectum could be repaired. In this case I first repaired the perineum and anal sphincter (after preliminary colostomy), and at a later stage attempted to close the whole vulval outlet. This procedure creates a "cloaca," making one compartment only of the rectum, vagina and bladder, with one opening, the anus.

This operation was almost successful at the 1st attempt, a small hole admitting a probe being left anteriorly. (The technique of this operation was shown me by my colleague Mr. T. H. Somervill, F.R.C.S., who had successfully treated other patients by this method. In his cases, however, the

anal sphincter had been intact.) This operation is mutilating, but is useful for such cases as these which cannot be relieved by any other means. This patient was a young woman, but had been abandoned by her husband, so that occlusion of the vaginal orifice was not a serious matter. In India there is no remarriage for women. If menstruation occurred, the flow would escape *per rectum*.

## Summary.

Vesicovaginal fistula, its causes, prevention and treatment are discussed.

A critical survey is made of the causes of failure in repair, of conditions which make repair impossible; and of the causes of death in patients treated by ureterosigmoidostomy.

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# Peroneal Palsy as a Complication of Parturition

BY

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## INTRODUCTION.

This condition was described by von Basedow[1] in 1838, following which the majority of the contributions to the literature came from French obstetricians. A comprehensive study of the available material was published by Hosslin[2] in 1905, who found that up to date only 81 cases had been recorded. During the last 2 decades, however, this complication of childbirth has received but scant attention from obstetricians; this has tended to give the impression that the condition is one of extreme rarity. Tillman[3] in 1935 reviewed the literature to date, collected 107 cases and added 8 more of his own. Recently Barns[4] reported on 3 cases in this Journal.

The 7 cases here described have been collected from the records of local hospitals over a period of 3 years. Cases 1 and 2 have been under direct personal supervision, Case 3 I saw after delivery and Cases 4, 5, 6 and 7 have been gathered from the records of St. Mary's Hospital, Manchester.

## CASE REPORTS.

*Case* 1, Mrs. E. D., aged 37 years, was a 2nd para who was first seen 1 week before her expected date of delivery. Her previous history was an interesting one. Two years before she had given birth to a living child weighing 9½ pounds. The labour had been extremely difficult, had lasted 36 hours and entailed the application of the forceps. Soon afterwards she had complained of severe pain, numbness and tingling in the left leg and foot, and noticed that she was unable to dorsiflex the left ankle. For the next 5 months she had been bedridden and quite unable to walk, but at the end of the 6th month had improved sufficiently to be able to get about, firstly on crutches and later with the aid of a stick.

During this pregnancy she had been under the care of her own doctor who, knowing the previous history, had decided to induce labour at the 8th month. She had been in a nursing home, but 2 attempts at medical induction had failed.

On examination in hospital the fundus was just below the xiphisternum and the vertex presenting in the right occipito anterior position. The head was high and mobile and there was a first degree of overlap.

The pelvic measurements were as follows:

Interspinous, 9½ inches; intercristal, 11 inches; external conjugate, 7½ inches; diagonal conjugate, 4 inches; intertuberous, 3½ inches.

She had, therefore, a narrow outlet with a narrow sub-pubic angle.

On walking, it was noticed that she dragged her left foot slightly, tending to walk on the ball of the foot. The measurements of the lower limbs were as follows:

Circumference of midthigh, right, 16 inches; left, 15¾ inches. Circumference of midcalf, right, 10½ inches; left, 10¼ inches.

Although these very slight differences are within normal limits it was quite obvious to the naked eye that there was wasting of the anterior tibial group of muscles on the left side.

Movements of all the joints were normal and equal with the exception of dorsiflexion which was weaker on the left side. The other ankle movements of extension, inversion and eversion were unaffected.

The reflexes were brisk and equal and there was no disturbance of sensation. This appeared to be the clinical picture of an almost completely recovered peroneal palsy.

As far as the 2nd confinement was concerned, a 3rd medical induction was successful. Labour progressed satisfactorily until the head was on the perineum when the application of the forceps was necessary. A living child weighing 8 pounds, 8 ounces was delivered without undue difficulty. Following this delivery there were no signs of peripheral nerve damage.

*Case* 2, Mrs. B. J., age 27 years, had been receiving antenatal care regularly from the 5th month of pregnancy onwards. Her pelvic measurements were as follows:

Interspinous, 8½ inches; intercristal, 9½ inches; external conjugate, 7½ inches; diagonal conjugate, 4½ inches; intertuberous, 4 inches.

One week before term the presentation was a vertex and the position right occipito posterior, with the head still high and mobile. She was admitted to hospital in labour and the head was then just engaging.

The 1st stage was normal, taking 15½ hours. Good 2nd stage pains commenced but after 3 hours there was no further advance of the head and no attempt at rotation. It was therefore decided to perform a manual rotation and forceps-extraction. The head was rotated with some difficulty and Neville-Barnes forceps applied. The blades slipped in easily and seemed to fit snugly—there was no necessity to reapply them. Traction was then applied in the usual manner but considerable force was necessary to produce any advance. The pull was a steady one, the blades did not show any signs of slipping and the vertex was delivered strictly in the antero-posterior position. A stillborn child weighing 9 pounds was extracted.

The following day the patient complained of pain, numbness and tingling over the outer aspect of the left thigh and the whole of the leg and foot. On examination it was noted that full dorsiflexion could not be obtained although there was no true foot-drop. Over the dorsum of the foot there was a diminution of epicritic sensation—this corresponding to the area where the paraesthesia was most marked. The symptoms gradually improved and by the 6th day she had regained full dorsiflexion and all the disturbances of sensation had disappeared. This sequence of events indicated a mild transient peroneal palsy.

*Case* 3. Mrs. R., age 20 years, was a primigravida who went into labour spontaneously at term and was delivered at home by her own medical attendant. The history was that she had been in labour for 2 days, the foetal presentation being a vertex in the right occipito posterior position. A manual rotation to a right occipito anterior position and forceps-extraction was performed. This was said to have been difficult and resulted in the delivery of a stillborn child of moderate size. The weight was not recorded. I saw her 10 weeks after delivery. Her external measurements were as follows:

Interspinous, 8¾ inches; intercristal, 9¾ inches; external conjugate, 7 inches; diagonal conjugate, 4½ inches; intertuberous, 4 inches.

On examination she limped badly, lifting her left leg in an exaggerated manner in order to clear the left toe from the ground. No obvious wasting was seen. There was a marked difference in skin temperature, the left leg being much colder and paler in colour. Movements showed that she had a complete loss of eversion and extension of the ankle joint. Sensation was normal to pinprick and cotton wool. The patient did not now experience any tingling in the leg but this had been marked over the outer aspect of the ankle for a few weeks following delivery. She was referred to an orthopaedic surgeon for treatment.

*Case* 4. Mrs. A. C., aged 34 years, a primigravida, had marked bowing of the long bones although there was no history of rickets. Her pelvic measurements were as follows:

Interspinous, 10¼ inches; intercristal, 11¾ inches; external conjugate, 8 inches; diagonal conjugate, over 4½ inches, intertuberous, 4 inches.

She was admitted to hospital in labour with head in left occipito anterior position and going into the pelvis.

She had a long 1st stage taking 77 hours and then, as good 2nd-stage pains lasting 2 hours had not caused the head to advance, the obstetrician in charge carried out a mid-forceps extraction. Neville-Barnes forceps were used and the delivery was exceedingly difficult. A living child weighing 8 pounds 2 ounces was extracted.

Later the patient complained of some pain in the right leg, and the following were found: There was a paresis of the anterior tibial and peroneal

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She had a long 1st stage taking 77 hours and then, as good 2nd-stage pains lasting 2 hours had not caused the head to advance, the obstetrician in charge carried out a mid-forceps extraction. Neville-Barnes forceps were used and the delivery was exceedingly difficult. A living child weighing 8 pounds 2 ounces was extracted.

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muscles of the right leg with a consequent foot-drop and the power of dorsiflexion was very weak. With massage and rest in the "anatomical" position there was a definite improvement but the foot-drop was still present when she was discharged from hospital. She was referred to an orthopaedic clinic for further treatment.

*Case* 5. Mrs. E. F., aged 38 years, had had 5 normal deliveries previously, the largest baby weighing 7 pounds. Her pelvic measurements were unfortunately not recorded in the notes. She was admitted to hospital as an emergency case, after being in labour for 44 hours—the membranes had been ruptured for 7 hours. She had had 4 vaginal examinations before admission and when seen in hospital the uterus was found to be at term with the head past the brim. Further vaginal examination showed the presentation to be a brow in the left frontal anterior position and the os was almost fully dilated. Under general anaesthesia attempts were made to convert the presentation to a vertex or failing that to a face, but the presenting part was so firmly fixed that all such attempts were of no avail.

The obstetrician thereupon performed a craniotomy and a dead foetus weighing 6 pounds 11 ounces was extracted. Two days later the patient complained of pain in the right leg and examination showed that she had weakness of dorsiflexion in the right ankle. The reflexes were normal and there was no disturbance of sensation. She was treated by massage and passive movements, and on discharge the function of the ankle had returned almost to normal.

*Case* 6. Mrs. C. C., aged 21 years, was a primigravida who had been receiving care at a municipal clinic. A few days before the expected date of delivery she was seen by the obstetrician in charge, who found that the vertex was still mobile and was presenting in the right occipito posterior position. It was decided that she should be confined in hospital. Her measurements were as follows:

Interspinous, 9¾ inches; intercristal, 10 inches; external conjugate, 8 inches; diagonal conjugate, over 4½ inches; intertuberous, 4 inches.

She went into labour spontaneously 1 week after the expected date of delivery. Some 15 hours afterwards the cervix was fully dilated, the head had made no attempt to rotate and the foetus was becoming distressed. A manual rotation was performed and Milne-Murray axis traction forceps applied. A living child weighing 8 pounds 3 ounces was extracted with considerable difficulty. The following day she complained of cramp-like pain in the left leg and shortly afterwards it was noticed that she was quite unable to dorsiflex or evert the ankle. On examination no sensory loss was detected. She was treated by rest in a right-angled splint and later by massage. Three weeks after delivery she still had an almost complete paralysis of the dorsiflexors of the left foot. The dorsiflexors of the toes were acting well. The invertors and evertors were acting poorly. Plantar flexion was strong. There was no sensory disturbance. She was referred to an orthopaedic clinic and lost sight of by the hospital.

*Case* 7. Mrs. L. G., aged 35 years, was a case of toxaemia of pregnancy occurring in a primigravida. She was referred to the hospital by her own doctor a month before the expected date of delivery as she had albuminuria and was vomiting persistently. The foetal head was beginning to engage in the left occipito posterior position. Her measurements were as follows:

Interspinous, 10 inches; intercristal 11½ inches; external conjugate, 7¾ inches; diagonal conjugate, over 4½ inches; intertuberous, 4 inches.

She was treated for the toxaemia by a low protein diet and rest, etc., but 14 days later albumin was still present in the urine and it was decided that labour should be induced. A Drew-Smythe catheter was used to rupture the membranes. Labour commenced and the vertex descended into the pelvis in the left occipito posterior position. The occiput made an attempt to rotate forwards and the 2nd stage was delayed with the head transversely fixed. There was some maternal distress and interference was indicated. With some difficulty the vertex was disengaged and rotated manually to the left occipito anterior position, the forceps applied and a living child extracted.

The following day the patient noticed tingling in the right lower limb and found that she was unable to dorsiflex or evert the right ankle. On examination it was found that she had a right peroneal palsy with no sensory loss. A right-angled splint

was applied and daily massage carried out. She went home 14 days after delivery and was referred to an orthopaedic clinic where massage and electrical stimulation were ordered. She was seen 1 month later and had improved considerably but still had a slight degree of paralysis. She failed to report again for further examination.

## Commentary.

Kleinberg[5] maintains that 1 or more of 3 essential factors are always present in this complication of childbirth. These are:

(*a*) Disproportion.
(*b*) Prolonged or difficult labour.
(*c*) Instrumentation.

The vast majority of cases reported do bear this out, but there are records in the literature of peroneal palsy after normal deliveries. The object of this paper is to attempt to correlate the position of the foetus with the side on which the paralysis occurs, in order to determine whether injury could be due to direct pressure on the sacral plexus or not.

It is easy to understand that with the greater diameter of the head lying in one or other oblique diameter of the superior strait of the pelvis, any adverse relation between these two dimensions might result in pressure on the sacral plexus (Hünermann 1892[6]). It is perhaps less easy to understand the vulnerability of the peroneal nerve component of that plexus—the subject of this commentary. The most likely reason can be based on pure anatomical *data*.

The peroneal nerve supplies the dorsal aspect of the lower limb (using the term dorsal in an embryological sense) and therefore arises from the posterior aspect of the sacral plexus. Thomas[7] writes "The upper roots of the sacral plexus do not lie on the pyriformis muscle but against the bony walls of the pelvis and are thus exposed to pressure during certain difficult labours. Dorsal offshoots of these nerves lie against the bone and receive the chief injury. The external popliteal nerve is made up of these fibres and therefore paralysis is chiefly located in the distribution of this nerve."

Thus the main mass of the sacral plexus is to some extent cushioned by the pyriformis, whereas the peroneal elements are devoid of any such protection. Linden[8] states that the condition must be rare in the flat non-rachitic or rachitic pelvis as the plexus is protected against pressure from the foetal head on account of the 2 deep niches on either side of the projecting vertebral promontory. On the other hand, in a generally contracted pelvis the paravertebral grooves are much shallower and place the plexus in a much more vulnerable position.

In Case 1 of the above series the presentation at the first pregnancy (when the damage was done) is not recorded. In the remaining 6 cases the presentations were as follows:

| Case | Presentation | Side of palsy |
|---|---|---|
| 2 | Right occipito posterior | Left |
| 3 | Right occipito posterior | Left |
| 4 | Left occipito anterior | Right |
| 5 | Left frontal anterior | Right |
| 6 | Right occipito posterior | Left |
| 7 | Left occipito posterior | Right |

In Cases 2, 3, 4, 5 and 6 the foetal head occupied the right oblique diameter of the pelvic inlet, therefore if direct pressure of the foetal head on the nerve were to occur the palsy should be a right-sided one. This was true in the left occipito anterior and left frontal anterior (Cases 4 and 5) but not in the right occipito posterior cases. Similarly in Case 7, the paralysis is on the opposite side to the diameter occupied by the foetal head. One can reasonably conclude that trauma due to foetal head pressure may be responsible in 2 of the 6

cases. It may also explain the cause of the paralysis in non-instrumental deliveries.

In order to explain the other 4 cases, however, one must presume that the peroneal nerve has been damaged by instrumentation.

## Summary and Conclusions.

1. Seven cases of peroneal palsy are recorded—3 from personal observation and 4 from hospital records.

2. These cases bear out the following points:

(*a*) The palsy follows difficult deliveries which entail extraction by the forceps.

(*b*) The paralysis is unilateral.

(*c*) It occurs on the side opposite to the one occupied by the greatest diameter of the foetal skull in the majority of cases.

(*d*) The cause in the majority of cases must be instrumentation. In the minority direct pressure of the foetal head may be the cause, though in these, too, instrumentation cannot be excluded.

(*e*) Pain and paraesthesia, though transient and not severe, precede the paralysis.

(*f*) The paralysis affects the dorsiflexors and evertors of the ankle.

(*g*) The paralysis will, in the majority of cases, clear up, and in those in which permanent damage is done the final paralysis is considerably less than the initial loss of function.

My thanks are due to Sir William Fletcher Shaw, Dr. F. H. Lacey and Dr. R. Newton for their kind permission to report cases. I am very grateful to Professor D. Dougal and Dr. C. P. Brentnall for their case records and also for their helpful criticism.

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## ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS.

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The following have been awarded the Diploma of the College:

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David Bradshaw Stewart.

# BOOK REVIEWS

"Gas and Air Analgesia," second edition. By R. J. Minnitt, M.D. (Liverpool), D.A. (R.C.P. and S. Eng.). Ballière, Tindall and Cox.

THIS is a small book written primarily for those interested in the relief of pain during labour, but a short chapter is devoted to showing this usefulness in the field of minor surgery. An appealing feature of the book is the obvious aim of the author to spare no effort in yet further improving upon present achievements, and this is particularly well brought out in the opening chapter which gives a short and interesting account of the evolution of gas and air analgesia.

The descriptions of the apparatus in its various forms, and the principles embodied, are clear and very well illustrated. Much space is wisely devoted to describing the apparatus in actual use upon the patient as the success of gas-air analgesia depends—and this is probably not fully realized by many—upon attention being paid to certain details. To stress this, commonly made mistakes are shown. A new technique, claimed to be a big advance on its predecessor, is described for giving better analgesia during the 2nd stage.

More space might have been devoted to the use of anaesthetic drugs with the gas-air analgesia apparatus. One is left wondering for what types of case this form of analgesia may be recommended —if, indeed, it may be safely employed in obstetrics at all, e.g., for the head sweeping over the perineum, or for repairing a torn perineum. The statement "the mother's pulse and foetal heart should be estimated hourly" is, however, a somewhat more serious fault: the foetal heart during the 2nd stage of labour (when the apparatus is more often in use) should, of course, be auscultated very much more frequently.

Apart from such minor faults this is an excellent little book which should be read by all—doctors, midwives and students alike—who practise obstetrics.

"Midwifery for Nurses," ninth edition. By Hendry Russell Andrews, M.D., B.S. (Lond.), F.R.C.P. (Lond.), F.R.C.O.G., and Victor Lack, M.B., B.S. (Lond.), F.R.C.P., F.R.C.S. (Edin.), F.R.C.O.G. Edward Arnold and Co.

THIS is, on the whole, a disappointing book. The illustrations, it is true, are good and particularly so in the case of that complex structure, the foetal circulation. Certain sections notably those dealing with abortion, normal and abnormal labour including the various mechanisms, are dealt with in a manner that warrants little criticism—though it is somewhat disconcerting to read that the doctor need be notified in a breech case in labour only if the legs, on vaginal examination, are found to be extended.

It is disappointing to find no mention of a blood-transfusion service (employing bank blood). Thus the nurse is advised in a case of severe antepartum haemorrhage when the doctor is delayed to send, on her own responsibility, the patient to hospital in an ambulance. Surely it would be wiser counsel to raise the lower end of the bed and inform, by telephone, the nearest maternity hospital so that an experienced member of the staff might proceed to the patient with all necessary equipment (blood-transfusion apparatus, etc.). It is even more reprehensible to advise the nurse, in a case of severe postpartum haemorrhage when the doctor is not present to undertake manual removal of the placenta with the patient in extremis (as an anaesthetic would not be required in such a case). An experienced and capable obstetrician would not undertake such a procedure nowadays without a preliminary blood-transfusion.

In dealing with the late toxaemias of pregnancy, too much stress is laid upon albuminuria and too little upon the blood-pressure changes. Further, whereas chronic nephritis—a rather rare concomitant of pregnancy—is specially mentioned as predisposing to late toxaemia, not a word is

mentioned of essential hypertension—a condition much more often associated with pregnancy and one which certainly favours late toxaemia.

A correct sense of proportion is totally lacking in many places. Thus the bogey of locked twins is unearthed, an unnecessarily large amount of space is devoted to insanity and to that extreme rarity, acute inversion of the uterus. One finds more space given to the admittedly interesting but actually unimportant condition known as pseudocyesis than to such an important and topical subject as diet during pregnancy. The space misspent in describing rarities should have been devoted to more profitable subjects. To give one example: prolapse should be mentioned as a very common late complication of labour which might be averted by properly performed exercises designed to strengthen the pelvic floor, and that it is the duty of the nurse in attendance during the puerperium to see that the patient carries them out. Naturally, a detailed account of one or two of these exercises should be presented to the reader.

In a book whose authors state that a principal aim has been to keep it as short as possible it is pointless and quite confusing to describe a similar condition in one place under the heading " flushed breast," and in another place under the heading " mastitis, or inflammation of the breast."

The chapters devoted to the infant are, on the whole, satisfactory, though mention might have been made when dealing with breast-feeding as to whether one or both breasts should be employed at each feed. On the other hand, the very scant reference to the problem of prematurity is a serious fault. Why, in the brief account that is given of the management of the premature baby, is no mention made of its admission as early as possible to a special nursery where life-saving facilities (oxygen, etc.) are available? Perhaps the defeatist attitude of the authors in their statement "it is unlikely that an infant of less than 32 weeks will live more than a few days" provides the answer.

In short, this is a book which, in its present form, I should hesitate to recommend to pupil-midwives.

" A Handbook of Gynaecology " for the Student and General Practitioner. By BETHEL SOLOMONS, B.A., M.D., F.R.C.P.I., 4th Edition. Baillière, Tindall and Cox.

THIS is a " cram-book " rather than a " handbook," and despite the fact that the preface states that the book has been written primarily to aid the student and general practitioner in his practical work and not to satisfy specialist needs, this is hardly the case. What justification is there for discussing, in some detail, the division of the cervix as a line of treatment in the chapters on Dysmenorrhoea and Sterility? The statement that the student must know about this operation for examinations is surely untrue, and furthermore can only do harm. If the student is led to believe he must know of this rarely-performed operation, he may well burden his mind with all the other rarities mentioned.

Another major fault is the listing of conditions under various heads, with little or no attempt to assist the student or practitioner in assessing their relative importance. Thus in a list of local uterine causes for dysmenorrhoea, an extreme rarity, viz., atresia of the cerviz, is sandwiched between two much more commonly found conditions, viz., acute anteflexion and hypoplasia. The introductory remarks to the symptomatology of fibroid uterus are even more remarkable: the reader is informed that the patient may suffer from severe toxic symptoms—which are dealt with in some detail—before any mention is made of the much more common symptoms. Again, although the " safe period " is discussed at great length it is disappointing to find that no mention of other forms of contraception is made: yet every general practitioner should have knowledge of them and be able to impart it to those couples who desire it.

While certain chapters, such as that of the routine examination of the patient and that of ectopic gestation, are written in an interesting and instructive way, the majority are written too concisely and contain such innumerable headings, divisions and sub-divisions—presumably to ensure that no point, however unimportant, is omitted—that by their very completeness they become distinctly boring. The concluding chapter by Dr. Chance dealing with X-rays and Radium in gynaecology is given in a most lucid and comprehensive way and deserves special mention.

For a book of its size too much space is devoted to operative gynaecology. Most will agree that the student should have some knowledge of common operations such as prolapse, but it seems point-

less to describe highly specialized types of operation, such as salpingostomy, in detail. It is surprising to find no mention of intravenous administration of glucose-saline in the treatment of post-operative ileus. Again, in the treatment of collapse following post-operative haemorrhage, one would have thought that blood-transfusion (employing bank blood) would have ranked more important than giving the patient plenty of hot drinks.

To sum up, this book has obviously been written from the examination standpoint. It will appeal to those students who like the facts presented in tablet form, but will be less appealing to the more thoughtful student and particularly to the general practitioner.

J. F. B. WYPER.

# REVIEWS OF HOSPITAL REPORTS

## REPORT OF THE GLASGOW ROYAL MATERNITY AND WOMEN'S HOSPITAL FOR 1943.

The Medical Report of the Glasgow Royal Maternity and Women's Hospital for the year 1943 follows its customary form. It is a most comprehensive publication. In the preface the general plan of the hospital service is outlined. There are 174 beds—73 antenatal, 75 lying-in, 15 for abortions, and 11 for isolation. In addition there are two labour wards with 9 and 6 beds respectively: in the isolation block there is a labour room with 1 bed. The hospital is divided into three units. The isolation wards are quite separate from these units. Frankly septic cases are removed from the hospital altogether to the isolation hospitals of the local authority.

Patients are mainly (85.5 per cent) drawn from the city of Glasgow. The remainder come from outlying districts which have no adequate hospital facilities; some of these are remote. Sixty-eight per cent of all admissions were considered abnormal. There were 4,794 admissions of which 3,361 resulted in deliveries. Being a large hospital the numbers are necessarily large. In a report this is an advantage for the statistical matter presented is less likely to be deceptive when reduced to percentages than is the case with smaller numbers.

In each section the condition under review is presented in varying degree of detail for the year concerned. Then follows a condensed tabular survey of the results of the previous five-year period. This latter is a valuable, if uncommon, feature.

Malpresentations are set out with single case data except in the case of breech presentation. Of particular interest in breech presentations are the results of the uncomplicated breech in primigravidae. (It would be helpful if the term uncomplicated were defined). There were 55 cases of uncomplicated primigravid breech. There were no maternal deaths, but 11 babies were stillborn, and 5 died. The foetal mortality is therefore 29 per cent. Prolapsed cord and cord presentation continue to exact the usual high toll of foetal life, 71.7 per cent of 46 cases. Of the 13 children which survived 3 were born spontaneously (as vertex presumably), 4 as breech presentations, 5 as breech following internal version, and 1 by the forceps. To a reader, attempting to assess the value of different lines of treatment, it would be helpful to know the degree of cervical dilatation when the condition was first diagnosed, but in no case is this stated.

Obstetric operations are classified and tabulated clearly. Some errors creep in, e.g., on page 30 we are told that labour was induced by artificial rupture of the membranes in 17 cases of contracted pelvis and on page 38 in 21 cases. The defining of 2nd stage delay (page 27) is erroneous and requires revision. Contracted pelvis and disproportion receive considerable attention, and merit careful study. Antepartum haemorrhages and the hypertensive toxaemias are set out case by case. In the accidental haemorrhage tables " first bleeding before delivery " is a heading. This might well be introduced into placenta praevia tables, if only to emphasize the importance of the " warning " haemorrhage. Similarly the " recording of symptoms and duration in days " (oedema, headaches, etc.) in the hypertensive toxaemias is valuable. It might be useful to add the nature of the earliest symptom or sign observed with the period of gestation at which it was first noted.

Perhaps the most outstanding feature of the whole report is the section dealing with maternal deaths. Factors sometimes overlooked are significant in the consideration of the numerical death-rate in any hospital. The number of maternal deaths in the whole area served by the hospital is obviously of importance, as also is the standard of practice in the antenatal clinics and of the practitioners in the district. Other factors too, the percentage of women who seek antenatal care, the standard of living of the community, may influence the hospital figures as do the more obvious facts such as the proportion of emergency to booked cases. While this is so 80 maternal deaths is a rather disquieting number. Viewed another way

it means that 1 woman dies in the hospital every 4.5 days. The causes of death are tabulated. One patient was not pregnant. Reviewing the details of the other 79 deaths, some emergencies had bad antenatal care, or none at all, only to die of hypertensive toxaemia. Some 15 patients died of postpartum haemorrhage. Of these 10 arrived in the hospital "shocked," "collapsed" or even showing "air hunger." If the transfer of a patient from her home to hospital means that her chance of recovery is thereby to be prejudiced, obviously the hospital service should be taken to the patient. One illustrative case will suffice.

"3257. Previous history, nothing of note. Obstetric history, three previous full-time spontaneous deliveries. Patient had had no antenatal care. Labour commenced at 1 a.m. on August 31st, 1943, and the district staff were sent for at 5.15 p.m. Hydramnios was diagnosed. A mature, normal child was delivered spontaneously at 8.40 p.m.; 10 minutes later the patient suffered a 3rd stage haemorrhage and lost 2 to 3 pints of blood. The district resident was called out and saw the patient, who was extremely restless and was bathed in a cold sweat. The placenta was retained. Morphine gr. ¼ and pituitrin ¼ c.cm. were given. On admission the patient *was in much the same state as when first seen but for slight air hunger* (italics mine). A plasma transfusion was immediately commenced and the condition of the patient improved thereafter. An unsuccessful attempt to express the placenta was then made and her condition deteriorated. One pint of blood was then transfused, but she died in the course of the transfusion. Postmortem not granted."

There is no mention in the report of the arrangements to cope with this kind of emergency. The value of an obstetrical "flying squad" is well known. An essential part of that team is an obstetrician of considerable experience: a house-surgeon whose obstetric experience varies between 3 and 6 months cannot be expected to have the clinical judgment necessary to deal with such a case entirely on his own. Expert preliminary treatment in the patient's home before she is removed to hospital may make all the difference between life and death. If the hospital does not have such a scheme the creation of one might well be considered.

Of 257 patients subjected to Caesarean section 11 died (4 per cent). In the Caesarean section tables we are not told, except when done for contracted pelvis, whether the patients were in labour, which were repeat sections, nor the type of anaesthesia used. Of these 11, 3 died of peritonitis, 1 of paralytic ileus, 1 of (spinal) anaesthesia, 1 of postpartum haemorrhage and the remainder from a variety of causes. There is fairly prevalent to-day the opinion that the mortality from Caesarean section, as a whole is 1 per cent. It has been suggested in the Royal Society of Medicine that, as the mortality has been reduced to 1 per cent, we should concentrate now on reducing its morbidity. Let us keep in mind Professor Claye's suggestion (*Proc. Roy. Soc. Med.*, 1943, xxxvi, 528), to institute a country-wide inquiry into Caesarean section. The lower segment operation, the ever widening indications, the free use of blood transfusion and chemotherapy have had a profound influence upon obstetric practice. The time is approaching when it will be possible, as it is now desirable, to institute this inquiry. What is the death-rate from Caesarean section to-day? It is said to be 1 per cent. Is it?

A tabular analysis of stillbirth (95/1000 viable births) and neonatal deaths (61/1000 live births), puerperal fever and puerperal pyrexia bring the year's report to a close. There is finally an appendix which gives a resumé of the hospital statistics for the past 10 years.

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## REPORT OF THE SIMPSON MEMORIAL MATERNITY PAVILION, ROYAL INFIRMARY, EDINBURGH, FOR 1943.

The Medical and Clinical Report of the Simpson Memorial Maternity Pavilion, Royal Infirmary, Edinburgh, for the year 1943 concerns 3,760 patients treated in the hospital and 784 treated in their own homes. The report is devoted mainly to the in-patient side of the obstetric service, but contains a brief summary of the cases delivered at home. There is also an extensive paediatric section.

The in-patient maternal section opens with a summary of mortality and morbidity. Then follow summaries of the usual complications of pregnancy and labour. These summaries are mainly tabular, headed in some cases by a brief explanatory statement or synopsis. In the case of breech

presentation, contracted pelvis, eclampsia, embryotomy and placenta praevia single-case data are given in more extensive additional tables. Three pages are devoted to short notes on maternal deaths. An interesting comparative table of the main features of this 1943 report and of that of each year back to 1930 is included.

There were 14 maternal deaths (one of these died undelivered) in the hospital and 2 patients died after being transferred to the City Fever Hospital, making 16 deaths in all. The total maternal mortality of the 3,760 in-patients is therefore stated as 0.42 per cent. If calculated in relation to live-births (to make it comparable to the national figure) there would be 16 maternal deaths for 2,754 live-births, i.e. 0.58 per cent. Some of the notes on these maternal deaths are just too short. One (booked) case (2410/43) of placenta praevia was treated by classical Caesarean section. " Death occurred immediately after the operation from severe postpartum haemorrhage. No autopsy." The presumption is that she was in a good state at the start of the operation. Was the uterus flabby all the time after extraction of the child? Did the surgeon have to consider whether hysterectomy was called for or not? Did this severe postpartum haemorrhage occur quite unexpectedly immediately after the operation was complete? What anaesthetic was used? We are not told. Another (booked) case 3,076/43 was considered to be normal until she had a third-stage haemorrhage which was treated by expression of the placenta and blood transfusion. Local uterine sepsis developed and, on the ninth day, secondary postpartum haemorrhage occurred requiring further transfusion. Intermittent haemorrhage recurred for another six days. The uterus was then explored and two small pieces of placenta were obtained. " Bleeding continued through the intra-uterine pack, and did not stop *even when this was repeated twice during the same day* (italics mine), so that ultimately subtotal hysterectomy was performed with continuous transfusion, but death occurred shortly afterwards. No autopsy." When was the pack first introduced—at the time when the two pieces of placenta were removed? Was it introduced first under anaesthesia? Were the repeat intra-uterine packings performed under anaesthesia? Did she have four general anaesthetics in one day? We are not told. The same lack of full information is tantalising in the summarized tables of the complications of pregnancy and labour. This is seen, for example, in the details given under Adherent and Retained Placenta. The placenta was expressed 24 times. Under anaesthesia? We are not told. The placenta was removed manually 31 times. Of these 55 patients 18 required blood transfusion, 32 had some degree of third-stage haemorrhage, while 33 were not admitted to the hospital until after the birth of the child. There is no means of telling, however, which patients required the blood transfusions. There were 18 cases of " failed forceps outside." The treatment in hospital was by expectancy resulting in spontaneous delivery, forceps, craniotomy, or Caesarean section: there was not one maternal death. But we are not told why the forceps had failed previously, whether the cervix was fully dilated on admission or whether there was any maternal morbidity following the successful treatment. I have stressed this aspect deliberately because by published results the trend of obstetric thought and practice is guided and influenced. Much time is spent and much labour expended in producing valuable reports such as this. But if they do not provide sufficient information for profitable study then, in measure, they fail in their purpose.

The sections set out with single-case data contain much of interest. There were 55 cases of uncomplicated primigravid breech delivery, resulting in 1 still-birth and 3 neonatal deaths (7.2 per cent foetal mortality). When twins, or one foetus of a twin pregnancy, are born by the breech " complicated " breech would seem the normal classification. Here they are included in the " uncomplicated " table. In this case the foetal mortality figure, however, is not materially altered by their inclusion.

The paediatric section is comprehensive. It is based on the infants born during the year, whereas the obstetric section is based on the admissions for the year. Neonatal mortality and morbidity are tabulated in some detail. Three deaths occurred in infants over 1 month old. The neonatal death-rate was 198 per 1,000 viable births for premature (viable) infants, 14.7 per 1,000 viable births for mature infants and 30.9 per 1,000 for all infants. No previable (under 2¾ lbs.) child survived. In fact none less than 3 lbs. in weight survived.

At the end of two weeks or previous discharge 83.3 per cent of infants were wholly breast fed, 5.9

per cent were breast fed with complementary feedings and 10.8 per cent fed entirely artificially. The causes of death have been investigated fully. Out of 108 deaths there were 101 autopsies. Of 143 stillbirths the cause was known in 64—determined by postmortem examination in 61 and by evident congenital defect in 3. The neonatal deaths and stillbirths are correlated with the normal and abnormal pregnancies of the mothers. Figures of the postnatal infant welfare attendances bring the report to a close.

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## THE SIXTH REPORT FROM THE DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY IN THE UNIVERSITY OF OTAGO.

### JULY 1942 TO JUNE 1944 INCLUSIVE.

The work was carried out in the Queen Mary Maternity Hospital, Dunedin. The students here each deliver 4 patients and witness the delivery of a further 20. After this each must deliver another 16 patients. This is an improvement of the minimal requirements for medical students in British Schools.

During these 2 years, exactly 1,000 women were delivered of 1,016 children, 16 of the patients having twins. The report makes pleasant reading. It might even be said to have a note of cheerfulness. Here we see midwifery as almost a physiological process, although occasionally something goes wrong. Only 73 of the patients delivered were admitted as emergencies; the other 927 all received antenatal supervision from members of the hospital staff.

There were 60 cases of toxaemia or hypertension. Some of these were severe, but the incidence of toxaemia on the whole is low. As far as can be judged the standard of blood-pressure adopted for hypertension is 140 systolic or 90 diastolic. At any rate one case is included with blood-pressure 150/80 and no other symptoms or signs. Of these 60, 4 had eclampsia—a fairly high incidence. No toxaemic patient died. There were 9 stillbirths and 2 neonatal deaths.

Malpresentation occurred 74 times, 34 as persistent occipito-posterior position, 2 as transverse lie, 3 as face and 35 as breech presentation. In the latter it is not possible to calculate the foetal mortality in uncomplicated primigravid breech delivery, but as there are only 10 primigravidae in this group it is not of great moment in this instance. Of the 35 breech babies one was stillborn at full-time as were also 3 of those born prematurely.

Delivery was effected by the forceps on 90 occasions (8.8 per cent), with 1 maternal death. Caesarean section was carried out 29 times. Eighteen per cent of the emergency cases were delivered by abdominal section while only 1.4 per cent of booked cases required the operation. It was indicated 17 times for disproportion, 3 for central placenta praevia, 2 for uterine fibroids and the remainder for a variety of indications. The lower segment operation was done 22 times and the classical 7, without a maternal death. Only 3 cases were morbid, 2 because of stitch abscess and 1 from thrombo-phlebitis. Three Caesarean babies were stillborn, the indication for these sections being eclampsia, accidental haemorrhage, and disproportion, with foetal distress. There were 32 cases of antepartum haemorrhage of which 12 were due to placenta praevia. Of the 9 cases of placenta praevia which did not require section, 2 were treated by Willett's forceps, 4 by puncture of the membranes while treatment was not required in the remainder. Postpartum haemorrhage occurred 7 times with 1 death. Hysterectomy proved necessary because of placenta accreta in 1 case in which delivery had been by the forceps. This case is also included among 28 cases of retained membranes (p. 14) which should probably read retained placenta.

There were 2 maternal deaths. Both occurred from shock following operative delivery. The first had haemorrhage from placenta praevia, manual rotation of the head and forceps delivery: postpartum haemorrhage necessitated manual removal of the placenta. In the other fatal case difficult manual breech delivery resulted in a complete tear. Death from shock followed 1 hour after delivery and repair. In the maternal morbidity table these 2 deaths appear to have been omitted. While recognizing that, the incidence of morbidity is very low. On the B.M.A. standard 10 cases occurred. In two years there was not one case of notifiable puerperal sepsis—a truly remarkable achievement.

Infantile mortality and morbidity are reviewed

briefly. Of infants born at full time 13 were still-born and 6 neonatal deaths occurred. The figures in this section (p. 20), however, do not add up correctly. Of 93 premature infants 15 were still-born and 6 died.

During the same 2 years there were 1,208 cases in the gynaecological wards. There were 11 deaths of which 5 were post-operative, 3 because of unrelieved carcinoma, and 3 from post-abortal sepsis. It would have been interesting to have had some details about the post-abortal deaths, but none is given. Once again the low incidence of some conditions is striking, e.g., there were only 2 cases of acute salpingitis, and 15 cases of sterility —in 2 years! Chronic cervicitis and downward displacements of the genital tract show a more normal frequency. Neoplasms both benign and malignant are found in customary frequency. Professor Dawson and his staff must be proud of these results for they are excellent. While in no way detracting from their achievement certain facts seem to emerge from this report. Their patients are no more immune to neoplasms, malignant or benign, or to the remote sequlae of obstetric injury than the average hospital patient. But they do appear to enjoy relative immunity to contracted pelvis, toxaemia of pregnancy, placenta praevia, puerperal infections and later pelvic infections and also perhaps sterility. It prompts the question of how far are climate, high standard of housing and food, economic and financial security responsible for the better results among these patients than among their less fortunate sisters at home.

ANTHONY W. PURDIE.

# INDEX TO CURRENT LITERATURE

In this Index an endeavour will be made, so far as war conditions permit, to maintain an up-to-date record of all titles bearing on obstetrics and gynaecology and the infant. The classification aims at making immediately available for the reader the titles under the various subject headings. In the majority of instances the title is obtained from the original publication. Where this has been impossible, the title has been obtained from the abstracting journal shown in brackets. Reprints will be welcomed and should be sent to the Editor.

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